NORTH CAROLINA Medical Journal

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December, 1952

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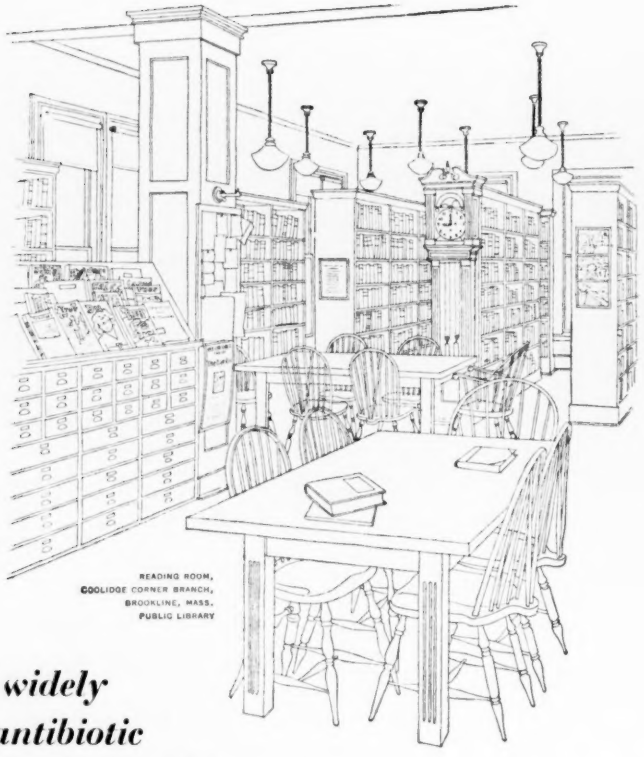

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THE BIGGEST HEALTH NEWS IN NORTH CAROLINA THIS YEAR OR ANY YEAR

SOME PEOPLE'S a great deal of walking around, and writing dull cubic footage figures in a notebook, but such routine duty often is spiced with adventure. Things

After an uneventful morning, he was reading a meter—with the aid of a flashlight — in a dark and dingy cellar. Hearing footsteps close behind him, he glanced over his shoulder, and his light caught the glint of a knife blade.

A WOMAN, with a bread knife in her hand, kept coming closer, as she muttered: "I'll kill you!"

The startled gasman gave the woman a shove, and scrambled to the basement door. Outside, the lady of the house, who was hanging up the wash, asked what was the matter. The gasman explained.

"Oh," she exclaimed in dismay, "it must be Nelly again. That's my sister. We put her away once, but I brought her home yesterday. We thought she was cured."

RECENTLY, in Kansas City, a gasman made a call on a new customer who told him to be a little careful as there was — phrased it—"a little wat basement." There cert

The gasman stepp stairs, and plunged i out, up to his neck. ceive any sympathy housewife? No, indeed. angry because he dripped all over her clean kitchen floor.

IT'S A FAIRLY common experience for a meter reader to be mistaken for someone else. One New York City gasman entered the basement of a home through the outside hatchway cellar entrance. He had just reached the meter when the lady of the house shouted from upstairs:

"Where do you think you're going, you big bum?"

Boards Sloop in River

The homecoming was mild compared with the pageantry when he returned last year to cremate his slain brother, formally assume own, and wed his queen,

miles down the Chaoer, the king's party left sh liner Meonia and the royal navy sloop

epping ashore on a specially elevated royal landing stage, he was greeted by Premier Pibul Songgram, government officials, and foreign diplomats.

Only Saturday Pibul Songgram had been restored as premier after a military clique forced his cabinet from office, set up a new hand picked parliament, and threw out the 1947 constitution.

Royal Decree Needed

Many observers have said the coup appeared partly aimed at curtailing royalist influence.

It is not known whether the king was officially informed of last week's events, which occurred while he was at sea, but a royal decree will be needed to legalize the new arrangement.

Pibul Songgram, resplendent in military uniform with a large sash, was the first to greet the king,

the formal speech o made by the king's br Chumphot, rather th premier or the head of the regen council, Prince Dhani Niwat.

The king thanked the welcoming committee and expressed wishes for the happiness and prosperity of his people .

The huge crowd cheered wildly

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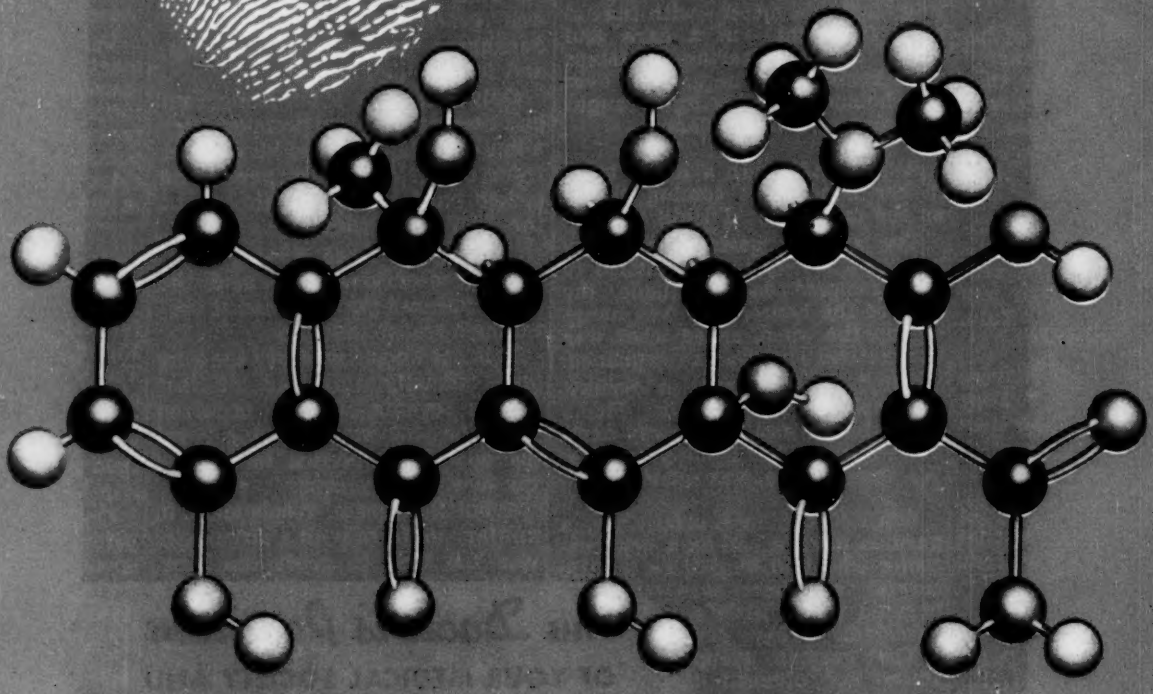

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Ward, E., Slocumb, C. H., Polley, H. F., Lowman, E. W., and Hench, P. S.,
Proc. Staff Meet. Mayo Clin. 26: 361, Sept. 26, 1951.

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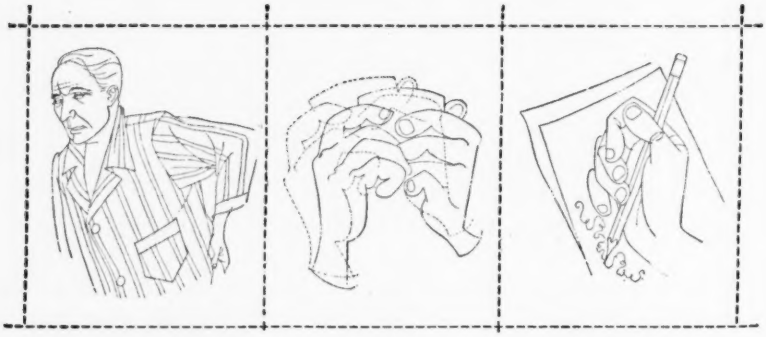

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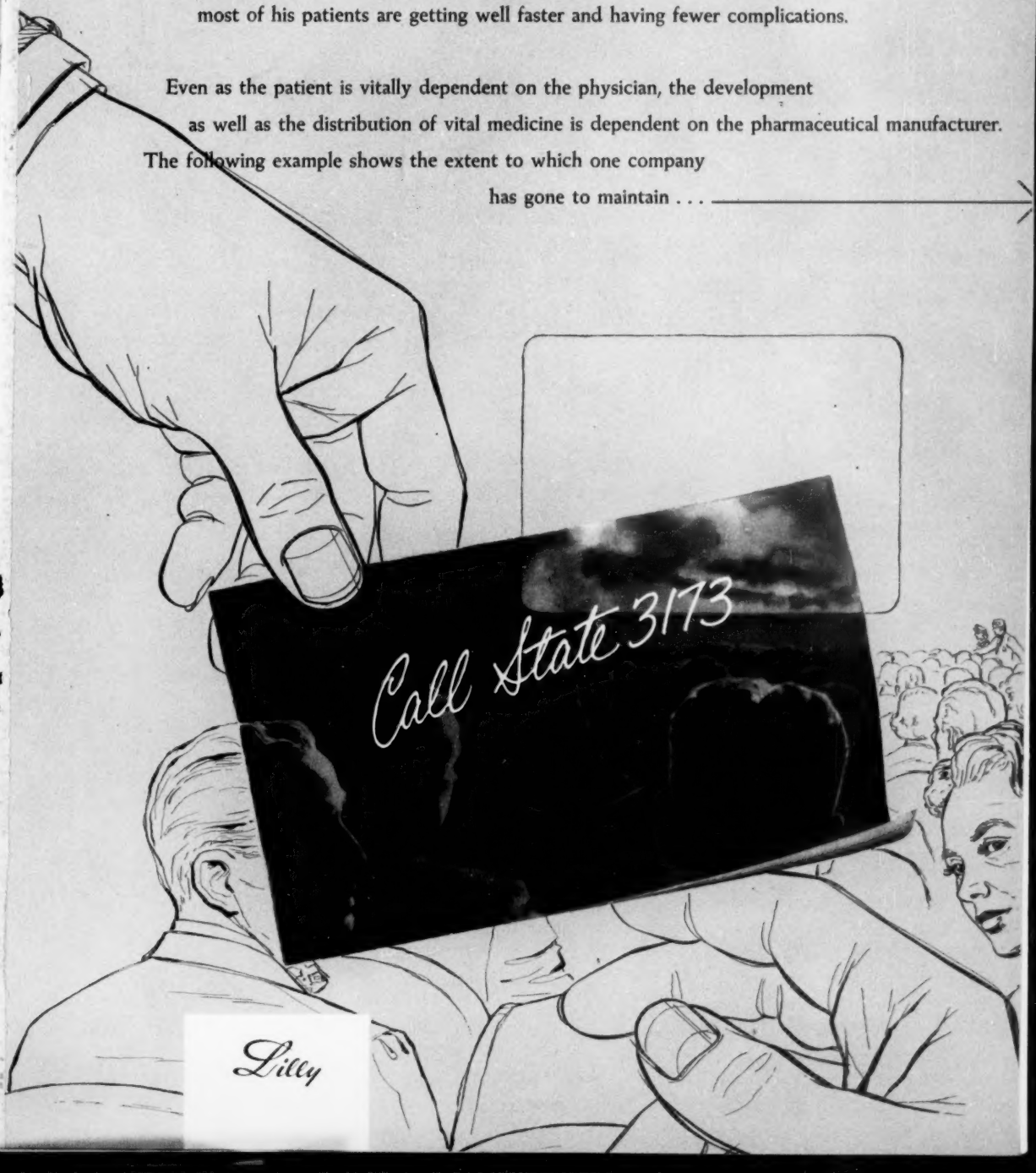

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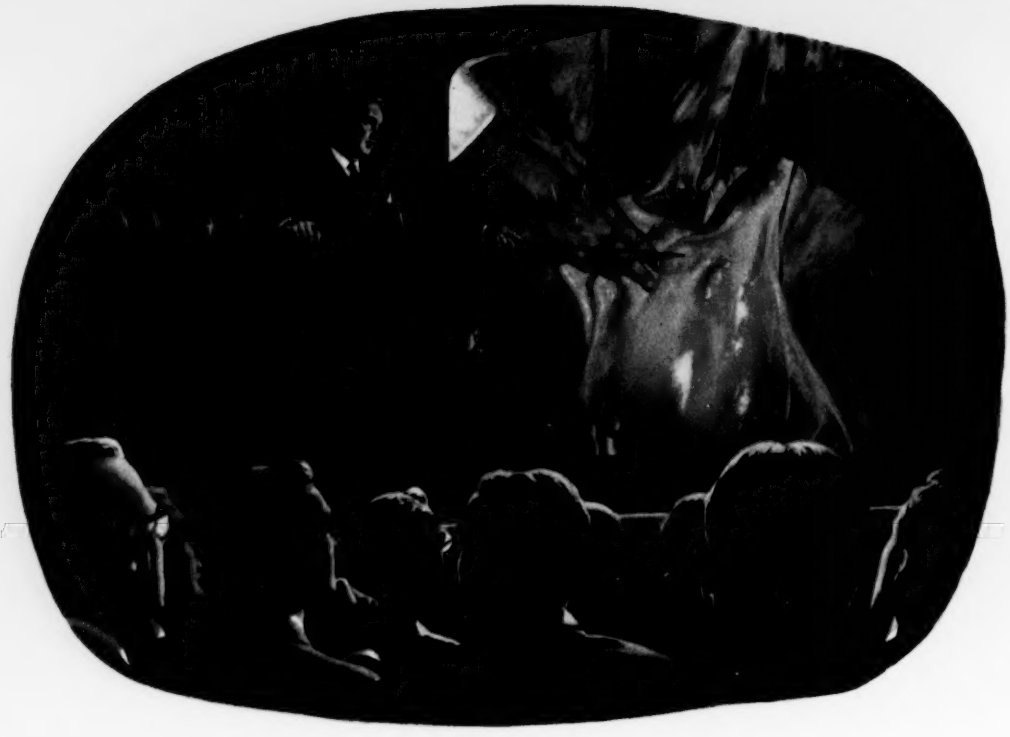

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NORTH CAROLINA MEDICAL JOURNAL

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VOLUME 13 DECEMBER, 1952 NUMBER 12

THE GROWTH OF SCHOOL CHILDREN AS AN INDEX OF UNDERNUTRITION IN THE COMMUNITY

A. HUGHES BRYAN, M.D.

CHAPEL HILL

In this paper we shall be concerned with plans to meet an emergency which we sincerely hope may never develop in any part of this country, or ever again elsewhere in the world: the problem of an inadequate food supply resulting from enemy action, with consequent serious undernutrition. As a part of civil defense planning, it is necessary to evaluate the means at our disposal of appraising the effects of a limited food supply upon the health of the population. The medical literature following the last two great world wars contains much pertinent information. It is not my purpose, however, to review this rather extensive body of knowledge, but rather to examine carefully one of the most useful indices of undernutrition—retardation of growth in children of school age.

In the event of a sudden and unexpected act of war, some parts of our country might suffer an immediate food crisis, to be followed later by a more or less prolonged shortage of food supplies. During this period—which might last for years, as it has in other countries—we can expect that food will be rationed and provision made to meet the nutritional needs of such priority groups as children, pregnant women, workers in essential industries, the sick, and the injured. In these circumstances the health officer must have at his disposal means for collecting objective evidence that all segments of the population are receiving food enough and of the right kind. The health authorities will be asked again and again whether or not strength, vigor, and the ability to wage total war are being undermined by an inadequate food supply or an inept rationing system. In gathering information with which to answer this important question, it will be necessary to employ more than one index of undernutrition and to take into account all facts which have a bearing on the question.

Read before the Section on Public Health and Education, Medical Society of the State of North Carolina, Pinehurst, May 7, 1952.

From the School of Public Health, University of North Carolina, Chapel Hill.

Means of Appraising the Effects of A Limited Food Supply

The experiences of other countries in previous wars indicate that the following lines of approach, if carefully followed, will lead to information of importance in assessing the effect on health of a limited food supply. In applying these methods it is well to remember that an evaluation of the changes revealed by repeated observations is often of more importance than the result of a single careful survey. Trends will demand our attention, and we will be asked whether the food situation is improving or deteriorating.

1 Food consumption studies. These consist of statistical analyses of the kinds and amounts of food consumed by representative samples of the population.

2 Medical evaluations. Under this heading are included such techniques as physical examinations to reveal the presence of malnutrition in segments of the population, studies of the prevalence of various diseases in the community, and an analysis of the causes which induce people to seek medical care. Biochemical studies of blood and urine may also be of assistance.

3 Measurements of height and weight. In the case of adults, trends in the average weights of representative samples appear to reflect changes in their caloric intake[1]. This

relationship may be disturbed if edema becomes prevalent. Children of school age respond to the privations of war with a decrease in their rate of growth, which is reflected in both their weights and their heights. This phenomenon of growth retardation, which was observed in many countries during both world wars, offers a useful and fairly sensitive index of undernutrition in the population. The remainder of this paper will be devoted to a critical study of this index.

The Effect of Undernutrition on the Growth of Children

A review of the literature on the changes in heights and weights of European children during the two world wars is contained in a recent comprehensive monograph on starvation[2]. Figures 1, 2, and 3 offer examples of the effect of undernutrition on the growth of school boys. Figure 1 presents graphs of the average heights and weights of 13 year old school boys in Oslo, Norway, for the period 1920 to 1947[3]. It will be noted that the years from 1920 to 1940 were characterized by a progressive increase in height and weight for age. This phenomenon, the secular increase in the growth rates of children, has occurred in many European countries and in the United States and Canada[4], and is attributed to the gradual improvements in food supply, medical care, and social and environmental factors which have taken place in those countries since the turn of the century. Following the invasion of Norway in 1940 the progress of this secular change was interrupted; the averages for height show little further increase in 1943, 1945 and 1947, while those for weights of 13 year old Oslo school boys were actually less in 1943 and 1945 than in 1940. This abrupt change in trends is to be attributed to undernutrition and perhaps to other adverse factors growing out of the German occupation of Norway.

The data presented in figure 2A are for boys entering school in Berlin at the age of 6 to 6½, and in figure 2B for boys leaving school at the age of 13½ to 14, during the years 1924 to 1933. In the case of the 6 year olds, (fig. 2A), we observe an irregular increase in average height from year to year, followed by a tendency to form a plateau from 1930 to 1932. That conditions were not ideal for growth is indicated by the fact that the over-all increase in average height from

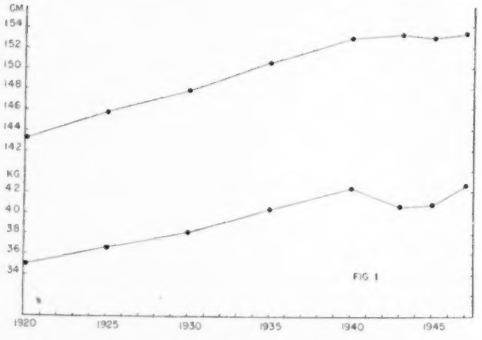

Fig. 1. Average heights and weights of 13 year old school boys in Oslo, Norway, from 1920 to 1947. Data from Strom[3].

1924 to 1932 was accompanied by very little increase in average weight—in other words, 6 year old boys entering school became progressively thinner during that time.

Turning now to the height and weight curves for boys 13½ to 14 years old, (fig. 2B), we observe a secular increase in both average height and weight from 1925 to 1930, followed again by a plateau for the next two years, 1931 and 1932. Finally, there is a slight upward trend in both measurements in 1933. (In interpreting this data it should be noted that the measurements of the children were made in the spring of each year and hence record influences on growth which occurred during the previous year or years.)

I believe that we see reflected in these year by year changes in the growth of 6 and 13½ year old boys the results of the widespread unemployment and economic dislocation in Germany which arose partly as the aftermath of the first world war, and were in part associated with the worldwide depression in the years following 1929. However, Wolff, the German physician who reported these results, offered quite another explanation[5]. The boys who were 6-6½ years of age in 1924, 1925, and 1926, and those who were 13½-14 in 1931-1933, were born during the period of most serious food shortage in Germany—namely, 1917 to 1919. Wolff attributes their decreased stature at age 6 to the effects of undernutrition on their mothers and on themselves during their prenatal period and early infancy. Furthermore, Wolff does not believe that the depression caused much change in the heights and weights of the Berlin school children whose

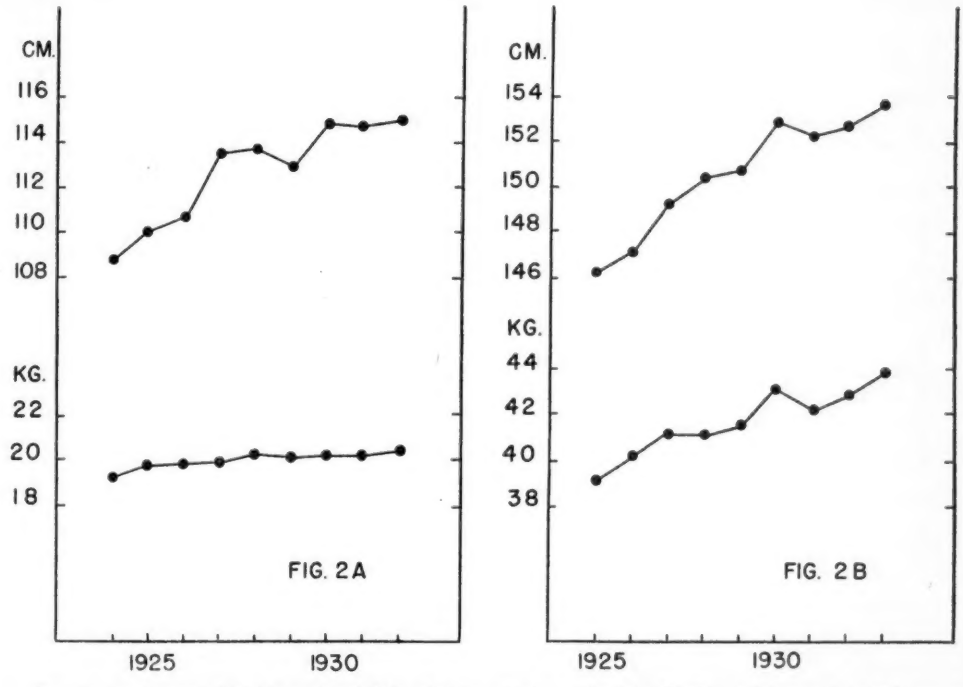

Fig. 2. A. Average heights and weights of boys entering school in Berlin at the age 6 to 6½ years from 1924 to 1932. Data from Wolff[6]. B. Average heights and weights of boys leaving school in Berlin at the age of 13½ to 14 years from 1925 to 1933. Data from Wolff[6].

measurements are presented in this article. The explanation that I have adopted, however—namely, that there was an irregular secular increase in height and weight up to 1930, followed by a leveling off due to the depression, allows us to fit Wolff's data into the pattern of retardation of growth which is easily recognized in other studies of the effects of undernutrition in children.

Finally, figure 3 shows the effect of undernutrition resulting from war on a sample group of Finnish school boys. We are indebted to Dr. Timo Jaaskeleinen for access to the original school cards of 144 boys, 7 to 13 years of age, who attended school in Reposaari, Finland, during the years 1935 to 1945. These cards record the results of periodic examinations and measurements during the school life of each child. We have selected for analysis only measurements made in the spring of each observation year in order to avoid the possible seasonal variations in growth[6]. Measurements are lacking for most of the boys in the springs of 1940 and 1943, and for this reason gaps appear in the graphs presented in figure 3.

Since the number of boys in the whole sample is small (144), the number in each class for each year becomes small indeed. For example, the number of 10 year old boys to be found in any one observation year is only 4 to 20. In order to avoid the irregularities which would occur if comparisons were based on such extremely small samples, we have utilized the statistical technique of covariance to calculate for each observation year a mean height and a mean weight based on all boys measured that year and adjusted to the age of 10½ years.*

*As used here, the covariance technique allows us to remove the effect of age on height and weight, and thus enables us to present adjusted averages of height and weight for each observation year which are based on all the data available for that year. However, covariance is not the statistical method of choice for a searching analysis of this data since (a) regressions of height and weight on age approach, but are not quite linear over the age span 7 to 13 years; (b) the variances of height and of weight are not homogenous over this wide age span; and (c) a cohort effect undoubtedly exists—children in the sample passed through the period of under-

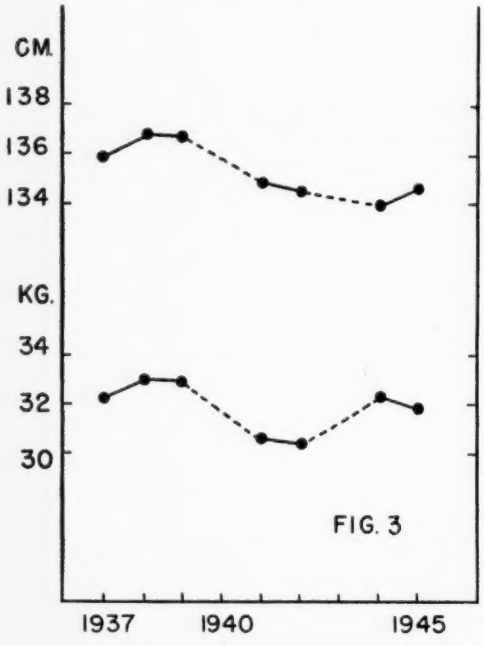

Fig. 3. Average heights and weights adjusted to an age of 10½ years (see text) of boys in school in Reposaari, Finland, from 1937 to 1945.

The food supply in Finland was normal in the years 1937-1939, although sugar became scarce by the fall of 1939. Rationing began in 1940, and 1942 was the year of greatest food shortages. Rationing continued until 1948, but the food supply available to the people became better and better during the years 1945 to 1948[7]. Figure 3 shows the effects of the resulting undernutrition on the adjusted heights and weights of a sample of school boys in Reposaari. The retarded growth of the war years is clearly demonstrated.

Growth Retardation as an Index of Undernutrition

The first point to be discussed is the extent to which factors other than nutrition might account for the growth changes noted above. It is known, for example, that groups of children from different socio-economic backgrounds vary in their average stature and weight, reflecting growth differences which may be attributed in part to differences in nutrition, but also to such factors as medical care, prevalence of ill health, and psychologic stresses. In our present state of knowledge it is difficult, in any given case, to assign relative values to the correlated factors which together condition the growth of children, and more research should be directed toward this end. In the meantime, the belief continues to receive wide acceptance that undernutrition is one of the most important factors in limiting the growth of children subjected to the stress of war.

The degree to which growth is interfered with appears to be in proportion to the severity of the nutritional defect. Thus the serious shortages which occurred in Germany and Austria during the first world war were associated with marked decreases in the average heights and weights of children[2]. More moderate changes in growth rates were observed in Oslo school boys in 1940-1945 (fig. 1); and in Britain, during the second world war, the still more favorable food supply was reflected in a general maintenance during the war years of pre-war standards of height and weight for age[8]. At most, decreases in average measurements occurred in 1940 and 1941 in some areas of Britain, mainly among older children, but the situation was generally rectified by 1943.

It is of interest to note that weight is a more sensitive indicator of undernutrition than is height, a quite general finding which is well illustrated in figures 1-3.

The importance of a base line, a period of control observations antedating the years of food scarcity, is evident. In many cases, such as that portrayed in figure 1, this control period exhibits a secular increase in average weight and height for age, and if the undernutrition is not too severe, its main effect may be to interrupt these secular changes.

Questions for further research

Finally, there is need for more research on the changes in the growth of children which occur as a result of the privations of war, including a more penetrating statistical analysis of the data already collected. Answers should be sought to the following questions:

1. If the food supply rapidly deteriorates, how soon can changes be detected in the growth rates of children? It would appear

nutrition, 1940-1944, at different ages and hence with different sensitivities to its effects. The second and third objections are more serious than the first. In collaboration with Dr. B. G. Greenberg we are carrying out a more suitable statistical analysis of the data kindly supplied by Dr. Jaaskeleinen.

that such changes might be observed during the first year of deprivation, but the matter requires more study.

2. At what chronologic ages are children most sensitive to the growth-retarding effect of malnutrition? The answer to this question would afford information of practical value in selecting samples of children for study with regard to the adequacy of the food supply during an emergency. We have found that children aged 8 to 11 show most clearly the influence of socio-economic factors on their growth[9], and our first assumption is that those ages might be the most suitable ones to study in a food emergency. In the meantime, further investigation of this point is indicated.

3. Are the effects of undernutrition shown equally by boys and girls, or do sex differences exist? In this paper, in the interest of simplicity, we have presented only growth data derived from the study of boys. Both boys and girls suffer a retardation of growth when subjected to undernutrition. The fact that girls mature at an earlier age than boys must be borne in mind, however; and, at some ages at least, the effects of food scarcity on growth rates appear to be less striking in girls than in boys.

4. Any given population of children passes through the years of deprivation at different chronologic ages. The immediate and more lasting effects of this period of malnutrition upon their growth may vary according to the sensitivity inherent in their age at the time of exposure. This phenomenon, termed the cohort effect by the statisticians, should be carefully studied in future investigation. A better understanding of the cohort effect would be of great assistance to those seeking to interpret changes in the heights and weights of children who have endured a period of malnutrition.

5. A further question for statistical investigation is concerned with whether it is better, for the purpose of detecting undernutrition in children, to study trends in their attained heights and weights, or to analyze increments in these measurements.

Use of the Index in Civil Defense

We now come to a very practical matter, the use of changes in the average heights and weights of samples of school children as an index to the nutritional status of a community during a more or less prolonged emergency. It has already been stated that this index would be only one of the means available to the health officer for observing the degree to which food shortages are undermining health. It should be used in conjunction with, and not in place of, such other methods as medical evaluations and food consumption studies. The index we are discussing is of particular value in emergencies of some duration, and has the advantage that the data on which it is based can be collected by other than trained medical personnel. It is taken for granted, of course, that the heights, weights, and ages of the children studied must be accurately recorded and the results analyzed by simple but adequate statistical techniques. The significance of small changes in average measurements must be accurately evaluated if the early effects of undernutrition are to be observed. This is a task requiring the combined efforts of health officers and statisticians.

It should be stressed that the index is concerned with *changes* in the average measurements of samples of children. This means that observations must be made on the same or comparable samples of children at regular intervals. It would be best to make these observations at the same season of the year in order to avoid seasonal variations in growth which would render interpretation more difficult. A base line of measurements of the same or comparable samples of children antedating the emergency will be necessary, especially since the effect of mild undernutrition may be only to interrupt the previous secular increases in group mean heights and weights.

Selection of subjects

The problem of selecting adequate samples of children for measurement will not be an easy one. Both race and socio-economic status modify the growth of children of school age[4]. In a community where the level of living varies from poverty to moderate wealth, and a mixture of races is found in the school population, the selection of suitable samples will require especial care. It must be remembered that the privations of war may not affect all strata of the community to the same extent. Some will suffer more, some less, and the delineation of the more vulnerable groups will be a matter of great importance in adjusting the ration to fit the needs of all of the population. Also, if trends in growth are to be studied, the same or comparable samples

must be available for examination at regular intervals, a consideration which further complicates the sampling problem. Fortunately, the determination and recording of height, weight, and age require little time; and the samples, therefore, need not be few or small.

In the event of enemy action we may expect migration away from areas of danger and into areas of comparative safety. The effect of these movements of groups of people will be to complicate greatly the sampling problem in some important areas. It will require considerable ingenuity to overcome or minimize this difficulty.

Interpretation of data

As the measurements are accumulated they must be interpreted; and to this task the health officer must bring not only the skills and knowledge of the statistician, but also a comprehensive understanding of the many factors in the local situation which may affect growth, as well as the accumulated experience of others who have studied growth and undernutrition in children. It will be important for him to recognize the first definite signs of retarded growth due to an inadequate food supply—when the situation has deteriorated to the extent that growth failure is marked, the shortage of food will have become so extreme as to be obvious to everyone. If well chosen, comparable samples of children show a significant decline in average weight for age, and particularly if they show also a decline in average height for age, it may be assumed that the ration is not adequate for these children. The probability of drawing a correct inference in such cases will be increased by taking into consideration other facts concerning the population and its food supply. While the presumption of undernutrition can be confirmed by the next series of observations of height and weight, one would scarcely suggest withholding remedial measures for such confirmation.

In many cases a negative result, the failure to observe a change in the growth rates of children when the food supply is limited, may be as important as a positive result. If food is scarce and rationing prevails, almost no one will be satisfied with his fare. Rumors of undernutrition will abound, and the morale of the community may well depend on a convincing and objective disclosure of the true state of affairs.

Summary

In the event of a prolonged food shortage occasioned by enemy action, questions will arise concerning the adequacy of the food supply to maintain health, vigor, and the ability to wage total war. One of the methods available to the health officer for assessing the extent of undernutrition in the population consists of the evaluation of trends revealed by periodic measurements of heights and weights of samples of children of school age. Retarded growth rates are presumptive evidence of an insufficient food supply or an inept rationing system, and this inference may be confirmed by the results obtained from other methods of studying the food situation and its effect on health. In this article are cited examples of the effects of undernutrition in limiting the growth of children. The use of such evidence as an index of undernutrition is discussed, and the need for further research along these lines is pointed out.

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Discussion

Dr. William P. Richardson (Chapel Hill): I would like to ask Dr. Bryan to comment on the advisability of accumulating data based on the information we now have to serve as a base line in the event of a restricted food supply.

Dr. Bryan: That is a good point, Dr. Richardson. We are going to need such data if a disaster occurs. Of course, much of our present data on weights, heights, and so forth is unreliable.

Dr. S. B. McPheeters (Goldsboro): In view of that comment, would you regard the weights and heights of children taken by school teachers as reliable data?

Dr. Bryan: In some cases I would, but in others I would question their accuracy. One would have to use the best data available.

Dr. A. H. Elliott (Raleigh): From your knowledge

of the study cited, what would you say about the effect of a limited food supply on teeth? Near the end of the depression, a dentist remarked that children had better teeth then than in normal times because they were unable to buy candy.

Dr. Bryan: That is an interesting point. Other observers have reported that in areas of malnutrition the incidence of dental caries, for one reason or another, is much lower than would be expected. I believe there are studies showing that in some countries there was less dental caries during the war than there had been previously.

Dr. William A. Fleming (Chapel Hill): May I ask Dr. Bryan about using adults for studies such as he has described? The advantages of using children, of course, is obvious.

In England during the last war the children were favored in every possible way. A considerable effort was made to serve an adequate lunch at school every day as a means of making up the deficiencies of the home diet. The children had many foods that adults could not get. Is there any way of complementing this index with others—using the weights of adults, for instance? Information bearing on production might be included.

Dr. Bryan: I am happy that you raised that point because it allows me to emphasize once again that this is just one index, and in the case of Britain, only one out of many.

Another point which I think is important is that people as a rule do not take kindly to food shortages and rationing, and many will claim to be suffering from an obscure type of malnutrition in order to get more food. Objective data will be useful in proving or disproving such claims.

As to the point that the weights of adults also decrease with undernutrition, there are figures on the decreases in weight that occurred in Germany and France. In order to get a true picture, however, it will be necessary for health authorities to carry out many different kinds of studies and to evaluate the health of the whole population, not only of school children but of adults in industry, pregnant women, and so forth.

In a war emergency the productivity of workers must be maintained. It is well known that if undernutrition becomes prevalent, productivity falls off, particularly in the heavy trades. In postwar Germany coal miners had to get sufficient food in order to carry on their work.

In conclusion, I would like to say again that this discussion covers only one aspect of an extremely complex question.

Child psychiatry: The average American child, as I see him in clinic or office, is a rather competent young organism. He has many problems: those of eating, dressing, going to school, getting on with siblings or peers; he has the problems of feedings, attitudes, relationships. In a sense these problems are his "needs"—are the circumstances of his physical and personal environment that demand his problem-solving attention. But I no longer leap to the conclusion that he must be taken on a psychiatrically conducted tour through this maze, lest he be lost or overwhelmed. He has a considerable capacity to digest these need situations that confront him; in so doing he creates the physical-personality structure that moves him on to the next series of problem-solving demands. None of us knows just what this is; call it "growth," or "Nature." The point is: the child ordinarily does what is demanded by the situation—Anderson, F. N.: Some Viewpoints on Child Psychiatry, California Med. 75:213 (Sept.) 1951.

NECROTIZING GLOMERULONEPHRITIS IN POLYARTERITIS NODOSA

Report of a Case

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Although it has long been recognized that arterial and arteriolar lesions in the kidneys occur frequently in polyarteritis nodosa[1], few instances of predominantly glomerular involvement are to be found in the literature.

Presented here is a patient with a long-standing, bizarre illness in whom a diagnosis of polyarteritis was made at autopsy. While the disease was in an apparently quiescent stage clinically, unusual necrotizing lesions developed in the glomeruli as a part of the terminal episode.

Case Report

First admission

The patient was a 30 year old white woman who was admitted to the North Carolina Baptist Hospital on July 31, 1951, because of anuria of 64 hours' duration. In March, 1951, she was first seen by one of us (J.C.W.) on admission to another hospital, because of weight loss, leg pains, pedal edema, and nonproductive cough.

The history revealed that in January, 1949, ankles, knees, wrists, and elbows had become swollen and painful, with episodes of exacerbation and remission. There were periods when all joints were involved, and other periods when only the ankles were painful. There was never any redness about the joints. By June, 1942, malaise was severe. A chest film at that time was reported as negative. In July, 1949, her last menstrual period occurred. During the next year she experienced periodic bouts of pain in the joints and lower legs, with increased weakness and loss of appetite. In November, 1950, photophobia with conjunctival injection occurred, and a nonproductive cough developed. Anorexia increased, with nausea and occasional vomiting. There had been no skin rash and no pleural or other pains than those of the joints. The

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From the Department of Pathology, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina.

Fig. 1. Roentgenogram showing the infiltration of the left upper and lower lobes at its height.

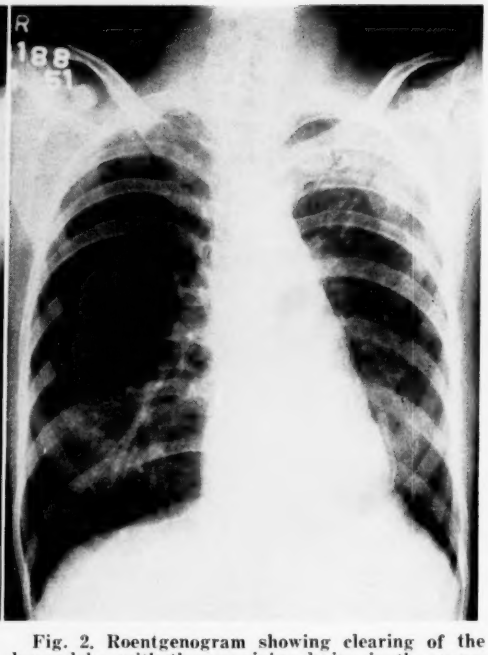

Fig. 2. Roentgenogram showing clearing of the lower lobe, with the remaining lesion in the upper lobe.

patient's symptoms gradually became more severe, with a weight loss of 20 pounds during the course of her illness.

On *physical examination* she was found to be chronically ill, emaciated, and pale. The blood pressure was 105 systolic and 70 diastolic, the temperature 100.6 F., and respiration 20. Circumcorneal injection, corneal vascularization, and small superficial corneal ulcers were present. There were signs of consolidation in the left upper lung field. No abdominal organs were palpated, and there was no tenderness. The vaginal and cervical epithelium appeared atrophic. Tenderness was moderate over the ankles and calves, and there was moderate pedal and ankle edema. The deep tendon reflexes were hyperactive in the upper and lower extremities. The Hoffman reflex was positive bilaterally and the Babinski positive on the left. Ankle clonus was sustained on the left and unsustained on the right. Vibratory sense was unimpaired.

Laboratory data: A review of the laboratory data was of interest in that white blood cell counts ranged from 11,000 with 73 per cent segmented neurophils and 4 per cent eosinophils to 9,400 leukocytes, with 69 per cent segmented neutrophils and 14 per cent eosinophils. The red cell counts ranged from 3,250,000 to 3,810,000 per cubic millimeter, and the hemoglobin level was 10.5 Gm. per 100 cc. The corrected erythrocyte sedimentation rate was 37 mm. per hour. Urine examination was consistently negative for albumin and red cells. The Kline test was negative. The serum contained 580 mg. of sodium chloride per 100 cc.; the carbon dioxide combining power was 49 volumes per 100 cc.; the nonprotein nitrogen was 37 mg. per 100, and total serum proteins were 6.8 Gm. per 100 cc., with 3.44 Gm. of albumin and 3.36 Gm. of globulin. The cerebrospinal fluid cell count, chlorides, protein, sugar, colloidal gold curve, and the Pandy test were within normal limits. Stools were negative for occult blood. A glucose tolerance test showed normal values.

An electrocardiogram was unremarkable except for a right axis shift. Roentgen examination of the chest showed an active process in the left upper and lower lobes of the lungs apparently not involving the apex. Numerous sputum smears and cultures for tubercle bacilli and for other organisms were done, with negative results. Bronchoscopy revealed thick, yellow, purulent material from the upper and lower lobes of the left lung. Smears

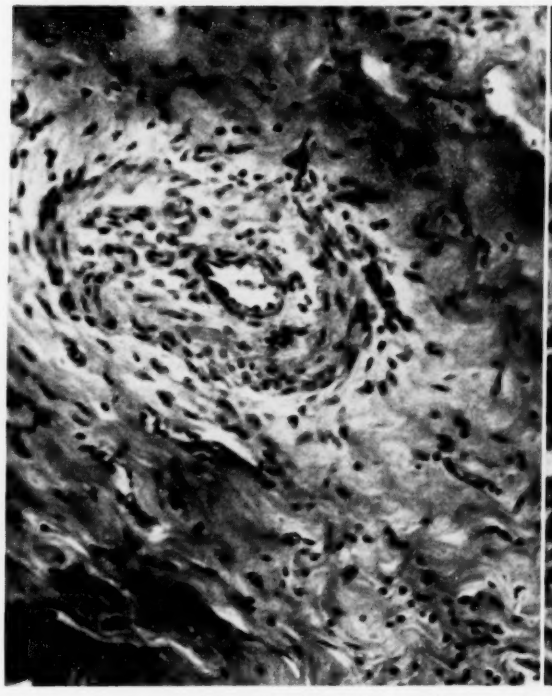

Fig. 3. An artery in the pericardium. The scarring and new vessel formation are seen to the left of the lumen.

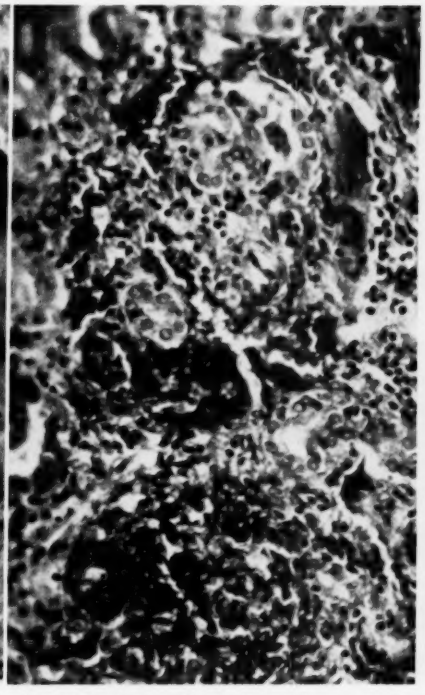

Fig. 4. Photomicrograph of the kidney, showing the extent of the changes. The black areas to the left represent fibrin in necrotic glomerulus.

and cultures revealed alpha streptococci; no abnormal cells. Numerous blood cultures were also negative.

Course of illness: The corneal lesions were treated by topical instillations of chloramphenicol, with good results. Therapy was nonspecific, with the administration of a high protein, low fat and high caloric diet. The pulmonary lesions cleared slowly (figs. 1 and 2), the low grade fever subsided, and a sense of subjective well-being developed, so that the patient was discharged after five weeks. During the succeeding three months she continued to improve, regaining her lost weight and to some degree her strength. Pedal edema and leg pains were gradually diminished.

Final admission

Approximately one week prior to her final admission and two weeks before her death, she began to have chilly sensations without fever, followed by abdominal pains and diarrhea. Ankle edema, dysuria and nocturia developed insidiously. There had been no preceding respiratory or other evident infection, change in habits, or exposure to toxic agents so far as could be determined. Sixty-four hours prior to admission anuria developed, and during the intervening period anorexia, drowsiness, and clouding of vision and mentality occurred. There was generalized itching but no eruption.

On examination at this time, the temperature was found to be 99.4 F., the pulse 88, and the blood pressure 110 systolic, 70 diastolic. She was chronically ill, dull, and hyperirritable, and had a uriniferous breath. Breathing was shallow. The keratoconjunctivitis had cleared, and the eyes showed no lesion. Her chest was clear and the heart was not enlarged. There was a harsh, grade II systolic murmur at the base. The abdomen was distended and tender, with a fluid wave and shifting dullness. No viscera were palpable. The tendon reflexes were hyperactive, and the Babinski and Hoffman reflexes were bilaterally positive.

Hemoglobin was 8.5 Gm. per 100 cc., the red cell count 2,850,000; the white cell count 11,840, with 87 per cent polymorphonuclear leukocytes, 5 per cent eosionphils, and 7 per cent lymphocytes. The nonprotein nitrogen was 172 mg. per 100 cc., the carbon dioxide combining power 1.13 milliequivalents per liter, chlorides were 90 milliequivalents per liter, and potassium 5 milliequivalents per liter. Electrocardiographic changes were thought to be consistent with hypocalcemia or hyperkalemia. Cystoscopic examination revealed edema and hyperemia of the bladder mucosa, but no obstruction of the ureters. Therapy was directed at correction of the electrolyte deficiences, but anuria continued and the patient expired on the seventh hospital day.

Postmortem examination

The body was that of an emaciated white woman somewhat older in appearance than her stated age. Edema was prominent in the lower legs and feet. One and one-half liters of clear straw-colored ascitic fluid was found. One liter of similar fluid was found in the left pleural cavity, 700 cc. in the right pleural cavity, and approximately 100 cc. in the pericardial cavity. Fluid found in the left pleural and pericardial cavities contained clotted fibrin. Fibrous adhesions were found over the posterior surface of the left lung, and over the right lower lobe. There were dense fibrous adhesions about the spleen. The heart was not enlarged and no valvular lesions were noted, but the myocardium was unusually flabby. The entire right lung and the left lower lobe were hyperemic, and both lower lobes were atelectatic. An irregular, wedge-shaped fibrotic area, approximately 3 by 4 cm. upon the surface, was found laterally in the left upper lobe. In this area of fibrous tissue there were several small cysts measuring up to 0.5 cm. in diameter, filled with pale yellow fluid. Irregular bands of fibrous tissue extended into the adjacent parenchyma along the indistinct borders of this area. There was no evident bronchial or gross vascular obstruction. No evidence of tuberculosis was found either in this area or in the hilar lymph nodes.

The spleen was not enlarged, but was surrounded by dense adhesions and the capsule was slightly thickened. The liver weighed 1,680 gm. and was hyperemic, with prominant lobules, but no other lesion. The adrenal cortex was thin, and the capsules appeared somewhat thickened.

The left kidney weighed 180 Gm. and the right 200 Gm. The capsules stripped easily, leaving unscarred cortical surfaces. Numerous pale areas the size of pinpoints were noted throughout the cut surface of the cortex with obscured cortical markings and a splotchy irregular reddish discoloration. The pelves and ureters were free of lesions, but the mucosa of the urinary bladder was hyperemic and edematous. The internal and external genitalia showed no gross abnormalities other than a slight decrease in the size of the uterus. The ovaries and fallopian tubes were approximately normal in size, and in each ovary several cystic follicles measuring up to 0.5 cm. in diameter were present. No gross changes were noted in the osseous system nor in the brain.

On microscopic examination no changes were found in the heart. The parietal pericardium was thickened, and the arteries beneath its pleural surface were surrounded by wide bands of hyalinized fibrous tissue through which were scattered lymphocytes, macrophages, and active fibroblastic nuclei. The vessel walls were eccentrically thickened and scarred, with prominence of medial nuclei. In some instances the intima was also thickened; the elastic laminae were generally disrupted. A few instances of new vessel formation (healed granulation tissue) were found in the adventitia. There was diffuse increase in fibrous tissue throughout both lungs, producing patchy thickening of the alveolar walls, more prominent in the left upper lobe. A few of the alveoli about the scarred area were filled with mononuclear phagocytes, and some of the smaller bronchi were dilated, with squamous metaplasia of the mucosa. Blood vessels in this area were generally thickened, and intimal proliferation was prominent. The inflammatory reaction was limited to a few scattered lymphocytes in the interstitial tissues.

In the liver there was some hyperemia and edema. The spleen showed no architectural change. There was no abnormality of pancreatic acini or islet structure, but occasional eccentric scarring of the medium-sized arteries was evident. Arteries in the submucosa of the gastrointestinal tract showed similar changes, and the perivascular connective tissue was often prominent and sometimes hyalinized with new vessel formation in the

adventitia. The zona fasciculata of the adrenal glands was thin, and there was little retained lipoid.

Widespread and profound changes involved the entire renal structure. The interstitial tissue was edematous, with infiltrations of lymphocytes, plasma cells, and mononuclear phagocytes. The glomeruli were similar in structure throughout both kidneys, were greatly altered, and in some instances were difficult to distinguish from the surrounding tissue. All glomeruli were the seat of varying degrees of necrosis, usually extensive, with fibrin thrombosis of the capillary loops and proliferation of the epithelium of Bowman's membrane. Proliferative cells filled the capsular spaces, surrounded the tufts, and in instances had themselves undergone necrotic change. Lymphocytic and plasmacytic infiltration in the glomeruli was prominent, as was the presence of mononuclear phagocytes. Fibrin deposition within the capsular mass had occurred in many instances in which capillary walls were obviously disrupted. Endothelial proliferation was active in most instances. Many of the afferent arterioles were involved in a necrotizing process but vascular injury was not evident otherwise. The tubules contained much cellular debris, precipitated albuminous material, and intact and broken red cells. The tubular epithelium was generally intact, but in some of the proximal convoluted tubules the cells were swollen and showed varying degrees of degeneration. Lymphocytic infiltration and edema were present in the pelves in moderate degree. The mucosa was edematous, with focal hemorrhages and lymphocytic infiltration. Although few of the arterioles of the muscularis were thickened and marked by prominent medial nuclei and perivascular scarring, the changes were not so marked as those noted in other areas. The endometrium was thin and atrophic, and the cervical epithelium showed foci of erosion and chronic inflammation. Vaginal epithelium was thin, with irregular edema of the basal layer, and lymphocytic infiltration of this layer and of the adjacent vaginal wall. In the ovaries numerous small cysts were lined by thin layers of flattened granulosa cells.

Comment

The nature of the renal changes and of the diffusely distributed vascular and perivascular scars presents the problem of interpretation. The renal lesions are not in themselves characteristic. That they are not primarily a manifestation of glomerulonephritis is suggested by the findings of vascular lesions in other organs. In the light of these vascular changes, it seems apparent that the two processes must be related.

The lesions of the blood vessels, with scarring, eccentric lumina, disruption of the internal elastic laminae, and healed granulation tissue, are the changes which are found in polyarteritis nodosa in its healed or healing stage. Remissions and exacerbations, with multiple system involvement, may occur in this illness; and occasionally an explosive recurrence may follow a period of quiesence.

In its earlier stages, the disease process in this patient had produced no complete obstruction of a major vessel and no infarct in any of the organs, but was manifested clinically as a peculiar, ill-defined disease characterized by arthritis, keratoconjunctivitis, mild fever, and a pulmonary lesion which resembled tuberculosis or some fungus infection. The urinary findings gave no evidence of renal involvement until the apparently abrupt onset of the terminal episode some two weeks before death, with hematuria of approximately one week's duration followed by anuria for eight days. The glomerular lesions were those of a destructive process developing terminally at the onset of the final illness, with proliferation of the glomerular lining cells, and necrosis of the glomerular tufts. It has been pointed out by Kay[2] that proliferative changes in glomerular tufts and in the capsules are evident by the sixth to eighth day after the injection of nephrotoxic duck serum in the rabbit, and that glomerular crescents are clearly evident by the eighth to tenth day.

A few instances of glomerulonephritis associated with polyarteritis have been described, among them the case of McManus and Hornsby[3]. This patient had a protracted clinical course marked by arthritis, pulmonary lesions, and a short terminal episode, much like that of our patient. The glomerular lesions apparently were basically similar to those of the present case. Daugherty and Baggenstoss[1] collected 7 cases of subacute and chronic glomerulonephritis associated with rheumatoid polyarthritis. The renal lesions developed more slowly and were of a more chronic or subacute nature than those of our case or those of McManus and Hornsby. Dav-

son, Ball, and Platt[5] noted in their series of cases changes in the glomeruli which were similar qualitatively.

As further evidence of the nature of this lesion, Herbut's report[6] may be cited. The patient was a 64 year old white man who was re-vaccinated for smallpox and who died some 37 days later of acute nephritis. The microscopic lesions were fundamentally similar to those of the patient under discussion. In addition, acute inflammatory and necrotizing lesions of the arteries could be found in organs other than the kidneys. They were considered by Herbut to represent polvarteritis nodosa, and he associated the vascular and glomerular lesions as manifestations of the same disease process.

It should be mentioned here that the usual renal lesions of polyarteritis nodosa are vascular, mainly arterial, and marked by necrosis and inflammation of the vessel wall, thrombosis and infarction, associated with secondary inflammatory changes in the parenchyma. Although the glomeruli are usually spared, they may be severely injured in recurrence of the disease process, as in the cited cases. This should be expected in view of their position in the circulatory system, particularly when experimental and clinical observations are considered.

Rich and Gregory[7] demonstrated the relationship in animals of the lesions of polyarteritis to hypersensitivity produced by injections of horse serum in rabbits. It is interesting that 7 of their 9 rabbits developed acute glomerulonephritis similar to glomerulonephritis in man. Since Lindemann's work in 1900[8], many others[9] have produced experimental glomerular lesions in animals by a variety of methods, the most successful being those involving sensitization. An exciting factor or demonstrable sensitivity has occasionally been found in instances of polyarteritis. The condition is sometimes associated with asthma or with a history of sulfonamide or other drug intake or sensitivity. In most instances, however, no such relationships are evident clinically, and in the present case no history of such factors could be elicited.

Summary

The case of a 30 year old white woman whose clinical course consisted predominantly of polyarthritis for about one and one-half years, slowly developing debility, low-grade fever, keratoconjunctivitis, and an exudative pulmonary infiltration in the left upper lobe, is described. Death was caused by acute necrosis of the renal glomeruli, manifested by hematuria, and followed by anuria for eight days prior to death.

The pathogenic features of this case, considered to represent an instance of polyarteritis nodosa with necrotizing glomerulonephritis, are reviewed.

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Discussion

Dr. John B. Graham (Chapel Hill): I would like to ask if there is any possibility of an antigen-antibody reaction in this patient?

Dr. Lee Large (Charlotte): I would like to ask Dr. Lide if he found vascular changes in the lung outside of the scar? The vascular lesions he showed were situated directly in a scarred region. As you know, Leibow has described pulmonary vascular lesions in scars.

Dr. Paul Kimmelstiel (Charlotte): Did you find widespread evidence of necrosis, or was this assumptive on your part?

Dr. Lide: In regard to Dr. Graham's question, we were unable to find historical evidence of an antigen-antibody reaction, either in the nature of a serum reaction or otherwise. We felt that some factor must have resulted in glomerular sensitization and severe necrosis, resembling the effects of anti-kidney serum in experimental animals.

In answer to Dr. Large, the pulmonary vascular lesions were entirely within the scar. They were not similar to the lesions in the arteries of other organs. There were intimal proliferation and fibrosis, and no medial scarring.

Dr. Kimmelstiel was interested in evidence of necrosis. In virtually every organ and area examined

we found scarred blood vessels similar to those which we showed in the slides. There were no active necrotic and inflammatory lesions outside the kidney. The changes were mainly those of the healed or inactive phase of polyarteritis.

A CASE ILLUSTRATING THE MORPHOLOGIC DIFFICULTIES ENCOUNTERED IN THE NEPHROTIC SYNDROME

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WINSTON-SALEM

The child whose illness and autopsy are discussed below presented no striking or new features. Clinically the case was considered a typical instance of the nephrotic syndrome progressing into renal failure.

In the discussion of this case in our pathology department, a variety of views concerning the undelying process was presented, and the clinicians were inclined to regard the pathologic findings as of little value in their understanding of the patient's course. It is this attitude, occasioned by what they consider the poor correlation between the clinical and pathologic findings in the nephritis-nephrosis group of renal diseases, which prompts the present discussion. The intimate relation between structure and function makes it likely that the gaps which exist between the morphologic and functional approaches will gradually be resolved. Morphology has pointed the way to many findings in physiology, and vice versa.

Case Report

The patient was a 4½ year old white girl who had 31 hospital admissions between the first, at the age of 18 months, and death three years later. When first seen she gave a history of infection of a foot and an ear about four months previously, and edema which had appeared two months previously. Examination disclosed anasarca, normal blood pressure, extreme proteinuria, hypoproteinemia, elevated serum cholesterol, and hematuria. There was no nitrogen retention and no anemia.

During the first year of observation she was frequently admitted for abdominal paracentesis and the treatment of various infections. The hematuria was so marked that surgical urinary tract disease was considered. Cystoscopy and retrograde pyelography were negative. Numerous urine cultures were sterile. During the remainder of her course nitrogen retention, acidosis, hypocalcemia, and tetany appeared, while the hematuria and proteinuria grew less marked. There was a transient response to ACTH in the form of diuresis. Death followed an upper respiratory infection. The highest recorded blood pressure was 120 systolic, 90 diastolic, during the last few months of life.

The gross autopsy findings consisted of the renal disease described below, together with myocardial hypertrophy (three times normal) chiefly left ventricular, chronic passive hyperemia of the lungs, spleen and liver, and effusion into all serous cavities. The kidneys weighed 45 and 40 Gm. respectively, roughly a 10 per cent reduction for this age group. Externally they showed prominent fetal lobulations. The capsules stripped easily, revealing a faintly pitted pale surface. The organs were firmer than usual.

On section the cortices were found to be narrowed and pale, the medullas pale, and the pelves were slightly more opaque than usual. There was no external obstruction of the urinary tract.

Microscopically, there was diffuse fibrosis and lymphocytic infiltration of the interstitial tissue, with generalized hyalinization of glomeruli. The latter process consisted of hyalinization of the glomerular tuft as well as of the capsule, and the former was usually more marked. No intact glomeruli were present. Proliferative or exudative glomerular lesions were not found. The tubules showed atrophy for the most part, although there were no "thyroid-like" areas. Scattered tubules containing protein casts were present. Some tubules showed epithelial ulceration with slight granulocytic reaction, and granulocytes were mixed with precipitated protein in some. A few areas of tubular regeneration were present. The subpelvic tissues showed changes similar to the interstitial tissue in general. Stains for fat and amyloid were negative. Masson's trichrome stain emphasized the degree of fibrosis.

Comment

The interpretation of these findings varied with different observers. I felt that they represented the end stage of chronic diffuse

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glomerulonephritis, with minimal terminal pyelonephritis. The hyalinization of the basement membranes of glomerular capillaries would be consistent with the presence of the nephrotic syndrome, but I do not think I would have made this association if the history had been unknown. Several of my colleagues thought the changes represented the end stage of "hematogenous" pyelonephritis; whether they took full cognizance of the history I do not know. Their opinion was based on the degree of periglomerular fibrosis, the marked tubular changes, and the degree of interstitial lymphocytic reaction.

We decided that someone should have the opportunity of looking at these slides unprejudiced by knowledge of the case, and I sent them to a former associate who is interested in this problem, Dr. I. R. Diamond of the Children's Medical Center, Boston. Dr. Diamond felt that the changes represented chronic diffuse glomerulonephritis, with the possibility of other, rarer conditions. He did not think that there was significant pyelonephritis nor that the more unusual forms of interstitial nephritis should be seriously considered. When told of the history he felt that this was the only feasible diagnosis.

These findings and opinions offer an example of why many people have remarked on the disparity between the clinical findings of nephrosis and the pathologic picture. Yet, clinically many people distinguish between a "pure" and a "mixed" form of nephrosis, the differentiation being based on the addition of renal failure, hypertension, and hematuria to the usual findings of the nephrotic syndrome in the latter.

There is increasing evidence that the nephrotic syndrome is one stage in a variety of degenerative renal diseases, characterized by the usual clinical features and probably presenting only one consistent pathologic finding, the presence of hyaline thickening of the capillary basement membrane and/or deposition of hyalinized material in the intercapillary space. This glomerular lesion varies in degree, and the variation probably accounts for the addition of features which clinically lead to the "mixed" appellation. The tubular changes are non-specific. Similar changes in the glomerular tuft occur in diabetic glomerulosclerosis (Kimmelstiel-Wilson's disease) and amyloidosis, and may be associated with the clinical nephrotic syndrome. Allen[1] claims that one may infer from inspecting the microscopic sections that the nephrotic syndrome existed. I would add to this statement the qualification that the kidney must be observed while the syndrome is present, since the later development of marked sclerosis may obscure the findings.

Ehrich and co-workers[2] suggest that the fundamental change in the nephrotic syndrome is in the glomerular basement membrane. They believe that glomerular nephritis and lipid nephrosis are distinct entities, with splitting of the basement membrane in the former, and irregular thickening of it in the latter. Lipid nephrosis may heal, or may be complicated by capillary thrombosis and/or exudation into the capillary space. The latter may lead to epithelial proliferation, "crescent" formation and late sclerosis. These complications may lead to renal failure and a terminal histologic picture indistinguishable from that of glomerular nephritis. This correlates well with many earlier observations. Ellis[3], for instance, found that patients with lipid nephrosis who did not die early, or recover, developed renal failure and a postmortem picture indistinguishable from that of patients with his type 2 nephritis. Ellis, however, felt that there was no fundamental difference between lipid nephrosis and type 2 nephritis.

A review of former observations in these conditions and a consideration of newer ideas obtained with newer staining methods make it apparent that the evolution of these lesions correlates well histologically with the clinical findings. It is true that the postmortem appearance of kidneys from patients with the nephrotic syndrome who died after the development of renal failure differs from that of patients who died of intercurrent disease. This fact is not an occasion for despair over morphology, however, but rather one for rejoicing. It shows the essential unity of these conditions, and the association of the nephrotic syndrome with a definite pathologic lesion which may heal or progress. It also demonstrates the relation of this lesion to other forms of renal disease. What causes the discontent is the fact that in any one case we may not be able to see the evolution of the process, and are called on to interpret what is essentially a scar. When many cases are considered together, there emerges in the light of their clinical course and chemical findings a fairly consistent picture, and one which points the way to further investigation

of the nature of the lesion in the nephrotic syndrome.

Summary

A case of the nephrotic syndrome, culminating with death in renal failure, is presented. The renal lesions suggested the end stage of chronic diffuse glomerulonephritis. The discussion attempts to emphasize that this is in keeping with current knowledge of the pathology of the condition and its histologic evolution, and does not indicate that the nephrotic syndrome is without morphologic correlation, as some have assumed.

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Discussion

Dr. George Margolis (Durham): Did you find in these kidneys any glomeruli which remained entirely normal, as far as you could recognize them?

Dr. Prichard: In all honesty I could not. All of them showed some degree of hyalinization and fibrosis, in the tuft and around it.

Dr. Paul Kimmelstiel (Charlotte): I would like to ask Dr. Prichard if Dr. Ehrich really claimed to have shown progression from the pure nephrotic stage. I had seen his exhibition some time ago, but I don't recall whether he showed the endothelial proliferation of glomerular nephritis.

Dr. Prichard: It is my recollection that he did. The essence of his concept is that the clinical nephrotic syndrome may be present with the pathologic changes of nephrosis alone which then heals, or such a pathologic picture may be complicated by the appearance of an exudative process which then brings about the pathologic features of glomerulonephritis.

Dr. Kimmelstiel: This case, therefore, is apparently primarily one of glomerular nephritis with a superimposed initial nephrosis? Or was it one of pure nephrosis to begin with?

Dr. Prichard: The original infection suggests glomerular nephritis. The clinical findings can be interpreted either way. The pathologic picture is not helpful. I really don't know.

Dr. Kimmelstiel: Lipoid nephrosis to me is identical with genuine nephrosis. The tubular changes are actually entirely secondary to the lipemia, and are only incidentally related to the nephrotic syndrome.

We can so wrap up a problem in a high-sounding phrase that it becomes lost to view. Sometimes, when we give a name to a disease or coin a term for a train of circumstances, there is a tendency to think the problem is solved. In the course of about 35 years of teaching, I have learned never to use a technical term without defining its meaning, unless I were foolish enough to wish to hide my ignorance from the students.—Murray, E. G. D.: The Emergency for Research in Unexplored Fields of Public Health, Canad. M.A.J. 66:276 (March) 1952.

PRIMARY HYPERPARATHYROIDISM MASKED AS PRIMARY RENAL DISEASE WITH HYPERTENSION

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DURHAM

Primary hyperparathyroidism was recognized as a disease of the bones years before its etiology was discovered. Historically, in 1891 a case of generalized osteitis fibrosa cystica was described by von Recklinghausen, who noted that in this disease the bones were soft and contained many cysts. In 1923, following the work of Gley and MacCallum and Voetglin, Hanson demonstrated an active principle in the parathyroid glands which would consistently elevate the serum calcium level when injected. In 1925 Collip extracted beef parathyroid glands in hydrochloric acid and obtained an extract which would cause a rise in serum calcium. The same year Mandl removed an adenoma of the parathyroid gland in a patient suffering from von Reckinghausen's disease and noted marked improvement in the patient's bone disease. Thus, hypersecretion of the parathyroid glands was identified as the specific cause of von Recklinghausen's disease, and the syndrome of hyperparathyroidism was elucidated.

The action of the parathyroid hormone, which chemically is identified as a large protein molecule with several amine groups, is not specifically known. However, administration produces the following changes: a rise in serum calcium; a fall in phosphorus; increased excretion of calcium, phosphorus, and nitrogen in the urine; increased calcium excretion in the feces; decreased ratio of nitrogen phosphorus excretion; a rise in the serum nonprotein nitrogen; and a late rise in plasma inorganic phosphorus paralleling the rise in blood urea after 24 hours[1]. Hypercalcemia and hypercalcuria in hyperparathyroidism are probably secondary to the extrarenal action of the hormone, since it has been shown that administration does not affect the rate of resorption of phosphate from the dog's renal glomerular filtrate[2], and since the resorption of calcium is greater, not

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Fig. 1. Parathyroid tumor and left lobe of thyroid gland.

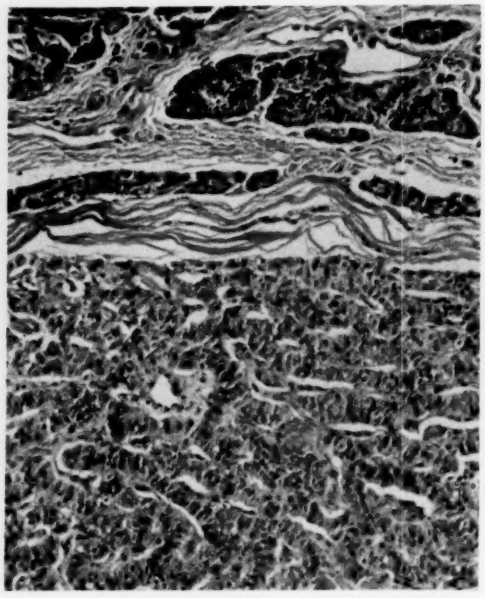

Fig. 2. Parathyroid tumor showing normal parathyroid tissue within tumor capsule. Hematoxylin and eosin. (x 145)

less than normal in hyperparathyroidism. The latter is presumably secondary to the increased levels of calcium present in the glomerular filtrate. A comprehensive discussion of the subject is given by Albright and Reifenstein[3].

A fatal case of primary hyperparathyroidism which clinically appeared to be a case of hypertensive vascular disease with renal insufficiency and cerebral complications is described in the following report.

Case Report

Eight months before death, a previously healthy 15 year old white youth began to have attacks of nausea and vomiting which lasted two weeks. Three attacks occurred before hospitalization. Two months before death he had a severe headache followed by unconsciousness and twitching of the right hand and face, and later by flaccid paralysis of the right arm. Fever and albuminuria followed. He then became semistuporous.

The blood pressure during his illness ranged from 180 systolic, 150 diastolic to 210 systolic, 150 diastolic, with a pulse rate of 80 beats per minute. Fundoscopic examination of the eyes was normal. Electrocardiograms showed no axis deviation. A leukocytosis of 13,900 was present, with a differential count of 65 per cent segmented polymorphonuclear leukocytes, 1 per cent stab cells, 9 per cent eosinophils, 1 per cent basophils, 8 per cent monocytes, and 16 per cent lymphocytes. The eosinophilia disappeared before death. Analysis of the urine showed the specific gravity to be fixed between 1.002 and 1.010, with 1 plus protein and 5-10 white blood cells per high power field in most specimens. Occasional hyaline casts and calcium oxalate crystals were seen. The nonprotein nitrogen serum level rose to 90 mg. per 100 cc., and the serum potassium fell to 2.19 milliequivalents per liter. The serum chloride fell to 76.2 milliequivalents per liter, while the sodium remained at 147.8 milliequivalents per liter. Calcium and phosphorus studies were not done on the serum. A mild anemia developed (hemoglobin 9.9 Gm.). Phenolsulfonphthalein renal excretion was only 12 per cent in six hours. Terminally, the patient developed a corneal ulcer of the right eye and died with aspiration pneumonia.

At autopsy, the body was found to be emaciated, and numerous petechial hemorrhages

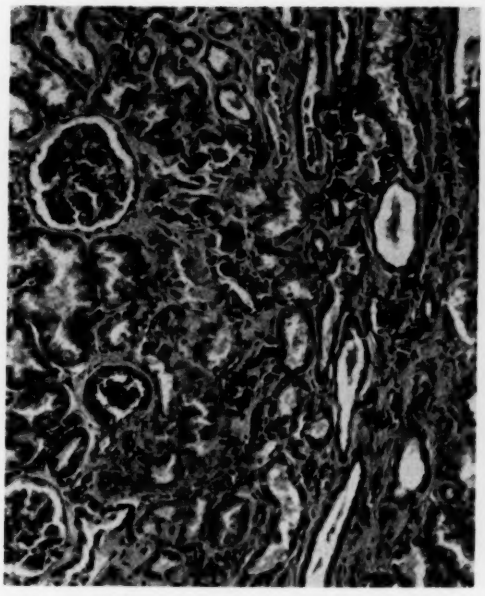

Fig. 3. Kidney showing calcification of tubular epithelium. Hematoxylin and eosin. (x 154)

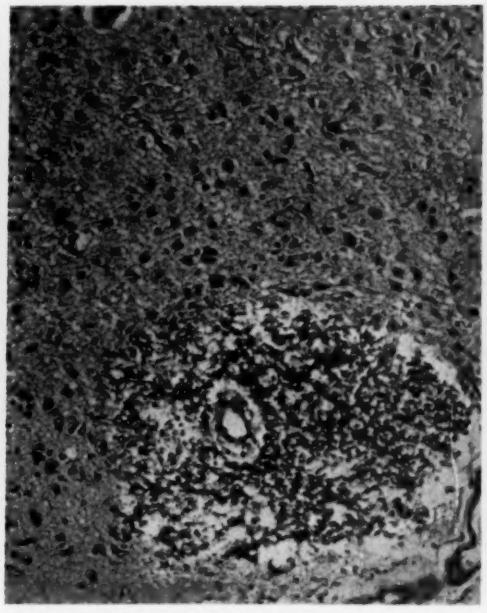

Fig. 4. Gliosis and perivascular mineralization of brain. Hematoxylin and eosin. (x 154)

were noted in the skin. Each lung demonstrated patchy areas of gray consolidation from 1 to 5 mm. in diameter in all lobes. The heart was dilated and weighed 200 Gm. The spleen and liver were passively congested. The kidneys were equal in size, weighing 150 Gm. each. Each cortex and medulla was swollen, and a fatty change was seen in the pyramids. No scarring was present. The thyroid gland was normal, but at the left lower pole a brown encapsulated parathyroid adenoma measuring 1.5 by 1 by 0.8 cm. was found. Four other masses of tissue, thought to be parathyroids, were found. Their combined weight was 14 mg. The combined weight of the adrenal glands was 10 Gm. All other endocrine glands and organs were normal. The brain weighed 1,500 Gm. and showed moderate dilatation of the entire ventricular system, but no obstruction. A 5 cm. focus of cortical encephalomalacia was present in the left parietal lobe.

Microscopically, the parathyroid adenoma consisted of cords and strands of proliferating uniform large cells which had abundant acidophilic cytoplasm and large oval vesicular nuclei similar to the chief or principal cells. A tubular pattern was formed by the cells where they surrounded vascular channels. Normal parathyroid tissue was compressed within the capsule of the adenoma. The three other parathyroids were not abnormal. The lungs and kidneys had numerous foci of metastatic calcification. Calcification was present in the alveolar walls of the lungs, and, in addition, there was an extensive, acute lobular pneumonia. The calcification of the kidneys occurred in all parts of the tubules but predominantly in the proximal convoluted and collecting tubules. Calcium casts occasionally occluded the lumen of the nephron, but usually calcification was limited to necrotic tubular epithelium. There was parenchymatous and fatty degeneration, with necrosis of the epithelium of the convoluted tubules. There was no calcification of the stomach wall. The bones did not show the lesions of osteitis fibrosa cystica. The brain had numerous bilateral microscopic foci of necrosis. In necrotic areas, there was marked proliferation of large gemistocytic astrocytes and microglia, with perivascular iron and calcium deposition. There were deposits of hemosiderin in the lungs, liver, spleen, parathyroid gland capsule, and brain. The bone marrow was hypoplastic. Striated muscle from rectus abdominus showed foci of necrosis.

Comment

Renal disease in primary hyperparathyroidism may be attributed[3] to three sets of pathologic processes. First, hypercalcuria and hyperphosphaturia create an increase in the osmotic pressure of the glomerular filtrate[4]. This produces polyuria and dehydration, which are followed by polydypsia. Second, the supersaturation of the glomerular filtrate results in precipitation of calcium salts, as calcium phosphate and calcium oxalate, in the calyces and pelvis of the kidney. This process leads to urinary obstruction, hydronephrosis, and pyelonephritis. Third, calcification occurs in the renal parenchyma proper, usually in the lumen of the collecting tubules but often in the necrotic or degenerating proximal convoluted tubular epithelium. This process may be due in part to the saturation of the glomerular filtrate with calcium and phosphate ions, but also involved is a primary injury to the renal tubular epithelium.

Following obstruction of the collecting tubules by calcium casts, proximal dilatation and epithelial degeneration occurs[3,4], resulting in a functionless nephron. Hypertension is a frequent accompaniment of primary hyperparathyroidism[3,5]. A low phenolsulfonphthalein excretion and a rising blood nonprotein nitrogen are associated with the degree of renal damage. This entire syndrome may often be observed in cases of acute primary hyperparathyroidism[3,5b], but the degree of renal damage and the marked hypertension observed in this case are not commonly observed.

Metastatic calcification occurred in this case in the lungs and kidneys, but not in the stomach wall. The lung and renal calcification observed here are classical[6]. The cerebral lesions are unique. When histologic study of the brain in primary hyperparathyoidism has been reported, the lesions have been described as hyperemia and perivascular hemorrhage. Hueper[7] reports cortical degenerative changes adjacent to the areas of hemorrhage in dogs given parathyroid extract. In our case, the cortical necrosis is more likely related to the hypertensive state. The cortical mineral deposits are probably secondary to the necrosis[8].

The absence of the classic bony lesions of osteitis fibrosa cystica in this case is not unusual. In cases of less than one year's duration[5b], this has been the rule. Although hypercalcuria and hyperphosphaturia occur, as long as the intake of dietary calcium is adequate there is no change in the bony mass[3].

Conclusion

Primary hyperparathyroidism may produce a syndrome in which the outstanding clinical facts point to primary renal disease. This syndrome may include hypertension, cerebral vascular complications of hypertension, and renal insufficiency.

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Discussion

Dr. Paul Kimmelstiel (Charlotte): I would like to ask Dr. Race if the changes in the glomerular vessels are sufficiently diffuse to account for hypertension, and, if not, how would he explain the hypertension in these cases.

Dr. Race: In some areas the renal vessels were slightly thickened, but in my opinion not to the degree usually seen in hypertension. The absence of glomerular lesions suggesting chronic glomerular nephritis led us to conclude that the vascular lesions were not there also. I can give no explanation for the hypertension other than widespread renal damage.

Dr. Kimmelstiel: You think the hypertension in this case is renal in origin?

Dr. Race: I do.

Dr. John Graham (Chapel Hill): Is not hypertension characteristic of the terminal stages of renal damage, Dr. Kimmelstiel?

Dr. Kimmelstiel: I think that if the kidney is the cause of hypertension, it is by means of impaired circulation, which should therefore require widespread involvement of either the vessels or glomeruli, or both. If that is not the case, one does not expect hypertension to occur, and it usually does not.

Dr. Graham: How about mercuric chloride poisoning?

Dr. Kimmelstiel: No, not to my knowledge.

Dr. Race: The papers of Muirhead and others report a controlled series of animals in which one group had bilateral nephrectomies and developed hypertension while being maintained on an artificial kidney. In another series, the kidneys were left intact and the ureters were tied, but despite nitrogen retention, and so forth, there was no hypertension. This would point to a humoral substance which is either being excreted by, or destroyed in the kidney.

Dr. Robert Prichard (Winston-Salem): Are you going to bring that point out when you publish the paper?

Dr. Race: No. We dealt almost exclusively with the hyperparathyroidism **per se.**

Dr. George Margolis (Durham): There are two things I would like to comment on. First, in regard to hypertension, I think others have shown that an agent which produces hypertension is retained. In the present case, I would like to ask your opinion of the reversibility of the process. If the parathyroid dysfunction had been relieved, would these kidneys have returned to normal functionally?

Dr. Race: I think there would have been some return because of the absence of vascular damage. The epithelial damage was quite widespread, but it certainly could have been repaired, with quite a large degree of functional recovery. I recall a series of patients with renal damage who had peptic ulcers and had consumed large amounts of milk and alkalis. When this was stopped, there was functional improvement.

TOXIC LESIONS ASSOCIATED WITH PARA-AMINOBENZOIC ACID

BERNARD F. FETTER, M.D.

DURHAM

In view of the administration of para-aminobenzoic acid as therapy for the collagen vascular diseases, it is felt necessary to add to the literature an instance in which changes in the heart muscle, liver, and kidney are possibly related to the administration of this medication.

Summary of the Literature

In 1949, Zarafonetis, Grekin, and Curtis[1] reported a series of 18 patients with various clinical forms of lupus erythematosus which were treated with para-aminobenzoic acid. In this series there were two deaths. One of these occurred in a child with subacute disseminated lupus erythematosus. She received sodium para-aminobenzoate for a period of 10 days. On the eleventh day she developed nausea and anorexia, and the drug was discontinued. The following day the patient expired, and postmortem examination revealed changes of an acute toxic hepatitis. The second patient who died was an 18 year old white female with disseminated acute lupus erythematosus who was treated for a period of six days. The drug was terminated seven days prior to death because of nausea and vomiting. At the time of postmortem examination the clinical diagnosis was confirmed; no mention is made of changes in the liver.

Read before the Section on Pathology, Medical Society of the State of North Carolina, Pinehurst, May 6, 1952.

From the Department of Pathology, Duke University School of Medicine, Durham, North Carolina.

In 1950, Upton and Zarafonetis[2] reported the histologic findings in rats subjected to prolonged para-aminobenzoic acid administration. These animals were sacrificed at one, two, three, and six months. No toxic effects in either the kidneys or liver of any of the animals were recorded.

The next pertinent report was added to the literature in 1951 by Cruickshank and Mitchell[3]. These authors report the finding in 3 patients who came to autopsy. The first of these was a child who had had rheumatic fever. Para-aminobenzoic acid was administered for a week, but was discontinued because of the appearance of red cells in the urine. After a period of two weeks the drug was again administered in similar dosages for a period of three weeks. At the time of autopsy there was extensive fat deposition in the myocardium and in the renal and hepatic cells. The second case reported is of a 10 year old girl, also with rheumatic fever, who was given the drug for a period of eight weeks. At the time of autopsy, in addition to the changes produced by the rheumatic fever, there was fatty infiltration of the liver and kidneys and myocardium. The third case reported was of a 13 year old boy who was treated for a period of 12 days. The findings at autopsy suggested one of the collagen vascular diseases. In this instance there was also infiltration of the heart, renal epithelium, and liver by fat.

Because of the changes presented by these last 3 cases, the authors decided to study the effects of the administration of the drug to rabbits. They were able to produce fatty alterations in the liver, kidney, and heart.

Case Report

In October of 1947, this 25 year old white woman manifested in rapid succession the following symptoms: (1) redness and tenderness of the fingers, (2) shooting muscular pains in the arms with soreness and stiffness of the arm muscles, (3) puffiness about the eyes and ankles. In a month the acute symptoms subsided, but there was residual soreness in the muscles and there developed a stiffness of the skin over the fingers and gradual wasting of the muscles of the arm and shoulder girdle. Liver function studies were negative.

In October of 1950, increased limitation of motion of the elbows and shoulders was noted on examination. She was given adrenocor-

Fig. 1. A section of the kidney showing extensive vacuolization of epithelial cells of the convoluted tubules. (Hematoxylin and eosin)

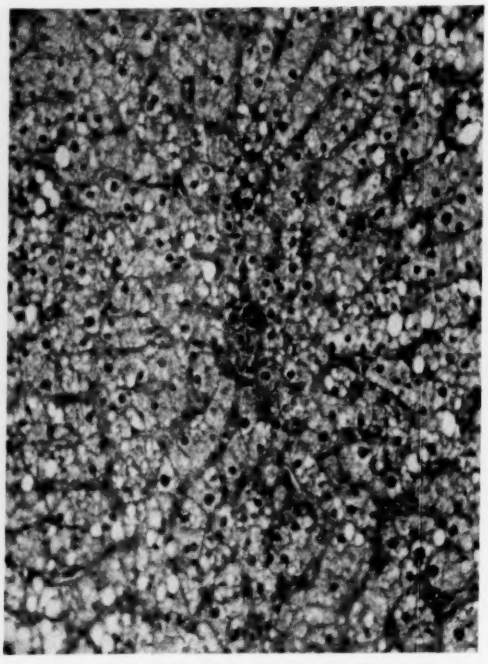

Fig. 2. A section of the liver revealing extreme vacuolization of all liver cord cells in field. Note central vein in the center of the photomicrograph. (Hematoxylin and eosin)

ticotrophic hormone and cortisone without benefit. She was seen again at the hospital in February of 1951, having difficulty in swallowing solid foods. Two weeks prior to death she was started on sodium para-aminobenzoate in the amount of 25 Gm. a day. This therapy was continued for one week, after which it was discontinued because of vomiting.

A postmortem examination was performed at this hospital approximately one and one-half hours after death. The over-all gross and microscopic picture was compatible with dermatomyositis. The following findings were considered unusual:

All sections of kidney showed an extensive vacuolization of almost all the epithelial cells of the tubules. Most of this vacuolization appeared to be basally situated. A stain for fat revealed that all these vacuoles were intensely sudanophilic. No remarkable alteration was seen in the blood vessels or glomeruli.

Sections of the liver showed extensive vacuolization of all the hepatic cells. This included both the cells in the central area and those at the periphery of the lobules as well. The vacuoles were large, and the liver cytoplasm was seen as a thin rim of eosinophilic material. A frozen section of the liver was likewise stained with Sudan IV, and all the vacuoles were found to be intensely sudanophilic.

The alteration in the heart muscle was not striking except that in focal areas there was vacuolization of some of the fibers. This vacuolization was also on the basis of accumulation of fat.

Comment

The preceding case is an instance of dermatomyositis which was treated with para-aminobenzoic acid. At the time of autopsy there were extensive fatty changes in the liver and kidney, and mild to moderate fatty changes in the heart. Toward the end of her course, this patient had difficulty in swallowing, and in considering any fatty alteration one must of necessity take into account the factor of nutrition. Combining this

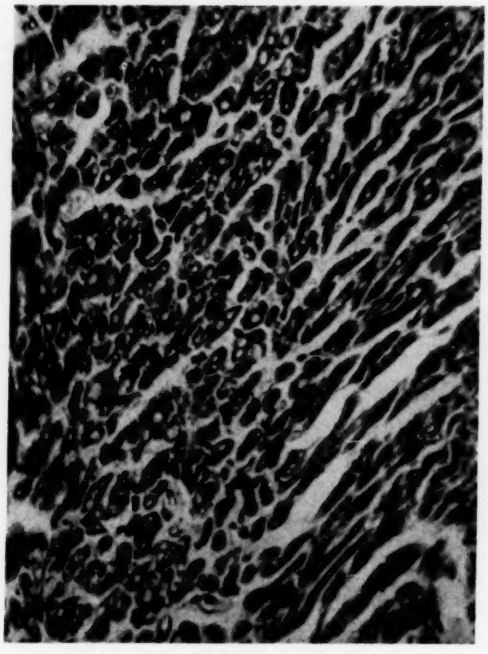

Fig. 3. A section of the heart showing vacuolization of many of the muscle fibers. (Hematoxylin and eosin)

factor with the presence of a generalized wasting disease, one might explain the fatty changes in the liver and heart. This explanation, however, is not sufficient to account for the fatty changes within the kidney, which resemble those seen in a toxic nephrosis. The fact that lesions of a similar nature have been produced in the heart, liver, and kidney of the experimental animal by the administration of para-aminobenzoic acid lends credence to the belief that this mechanism is at least in part responsible for the changes which have been found.

It may be that the administration of the para-aminobenzoic acid is solely responsible for all the changes which were observed. Such a conclusion, however, is questionable in view of the progressive, fatal disease process from which the patient suffered. This case, then, is presented as an instance in which the administration of this drug was probably responsible for a histologic alteration in the body tissue.

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Discussion

Dr. Robert Prichard (Winston-Salem): Was the patient anemic?

Dr. Fetter: Yes, she had a slight anemia, not marked.

Dr. Thomas Wilson (Raleigh): Was the relative dose of para-aminobenzoic acid excessive over a long period of time?

Dr. Fetter: It was given for one week, 25 Gm. per day of the sodium salt, which is not considered excessive.

Dr. Parker Beamer (Winston-Salem): The heart, I remember, was smaller than normal. Were the liver and kidneys enlarged grossly?

Dr. Fetter: The liver was enlarged, weighing 1700 Gm. The kidneys weighed 165 Gm. each.

Dr. Beamer: And that in a woman weighing 100 pounds?

Dr. Fetter: Yes.

Dr. Paul Kimmelstiel (Charlotte): What did the patient die from?

Dr. Fetter: She died from pneumonia.

Dr. Kimmelstiel: Was it sufficiently extensive to be fatal?

Dr. Fetter: As far as we know, it was not. Terminally, a trace of protein was found in her urine for the first time.

We are beginning to recognize that antibiotics are not uniformly without their dangers and disadvantages, great as have been the benefits their use has conferred. This is not simply a clinical therapeutic problem of individual cases, but has public health significance. Witness the interference with the production of immunity in typhoid fever cases treated with chloromycetin, resulting in an unprecedented frequency of relapses and retained susceptibility to reinfection, without any reduction of the carrier rate. Its extensive use in an outbreak of typhoid might well give rise to unprecedented problems in the management of the public health situation. Another type of antibiotic problem comes from the justifiable and intensive use of an antibiotic to treat one kind of infection, thereby promoting the development of another and different kind of infection. This is illustrated by the development of serious mycotic disease, for example, when aureomycin is used extensively to treat a susceptible bacterial infection. Indiscriminate use of antibiotics is also inducing astonishing changes in regional bacteriology which are at times detrimental to the patient.—Murray, E. G. D.: The Emergency for Research in Unexplored Fields of Public Health, Canad. M.A.J. 66:277 (March) 1952.

The major emphasis of the tuberculosis campaign has been directed to the prevention of the spread of bacilli; yet it was a startling fact that from 50 to 90 per cent of adults become and remain tuberculin positive even in those communities which enjoy a highly developed public health program. Mortality rates have been decreasing much faster than infection rates.—H. R. Edwards, M.D., The Am. Review of TB, March, 1952.

ELECTROCARDIOGRAPHY IN GENERAL PRACTICE

DAN N. STEWART, M. D.

HICKORY

As soon as a new technique in diagnosis or treatment has been devised, tested, and proved to be valuable, it is the duty of the general practitioner to incorporate it into his own practice whenever possible. It has been truly said that a general practitioner is a general specialist.

If the procedure is too difficult or if it requires expert knowledge, it should remain the function of the specialist. The greatest difficulty that confronts the general practitioner is to select among his patients those who might require a detailed blood examination, others who need a basal metabolism, and still others who should have x-ray visualization of the genitourinary tract. This entails a knowledge on the part of the physician of the *kind* of information that might be revealed by the specific type of examination.

In applying these ideas to the question of electrocardiography, it is necessary to know the various clinical conditions in which this method may be of help in diagnosis or treatment. Although more and more physicians are doing their own cardiographic work, usually they will need to call on some other physician to obtain and interpret their tracings. The main question, therefore, is: When is an electrocardiogram needed? It is an axiom that the more one knows of heart disease, the less he needs an electrocardiogram. Conversely, the more one knows about electrocardiography, the less he is dependent on it.

Conditions in Which Cardiography is Useful

In general, there are three main groups of cases in which the general practitioner may find electrocardiography helpful: (1) *acute coronary thrombosis* and *chronic coronary artery disease*; (2) all types of *cardiac irregularities;* (3) an *infection* where the possibility of rheumatic fever is present. There is also a miscellaneous group of less common conditions where tracings may be indirectly helpful.

Coronary artery disease

Electrocardiography probably has its greatest value in the group of coronary artery diseases. So many patients with coronary sclerosis, angina pectoris, and even myocardial infarction show nothing abnormal on ordinary physical examination that laboratory methods are often required to establish the correct diagnosis.

In making the diagnosis of acute occlusion or of angina pectoris, the history is all important. If the typical story is obtained, regardless of how normal any test or examination may be, the diagnosis of angina pectoris must be made, unless the patient is believed to be malingering. There are many instances, however, where the story is atypical and where the distress might be due to gas, gallbladder disease, or many other conditions. If the diagnosis of angina pectoris or coronary occlusion is certain, the physician does not need an electrocardiogram. He should obtain a tracing, however, when the diganosis is in doubt. By no means, however, does a normal electrocardiogram rule out either angina pectoris or coronary occlusion. A normal electrocardiogram does not guarantee a normal heart. The examination is valuable mainly when it is positive. However, several normal tracings taken during the first one or two weeks following an acute attack do point strongly against the possibility of myocardial infarction.

The electrocardiogram can only show evidence of myocardial damage. This may be due to coronary artery sclerosis without an acute coronary episode. On the other hand, there may be acute coronary disease, slight or marked, without a myocardial infarct, and in these cases the electrocardiogram may be unchanged. Thus, it is seen that the electrocardiogram may be most useful in finding disease in a heart that otherwise appears innocent. It therefore behooves us to use the electrocardiograph rather freely in patients suspected of coronary artery sclerosis, especially in males more than 40 years old with atypical symptoms.

There are a variety of changes that may help in the diagnosis of coronary artery disease. The most typical finding is an abnormal or inverted T wave. Frequently an intraventricular conduction defect will be discovered, and occasionally some degree of auriculoventricular block is present.

The physician should know that some changes may be due to old and inactive lesions that have nothing to do with the present

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complaint, and also that alterations may result from causes other than heart disease, such as digitalis, pulmonary embolism, vitamin B deficiency, myxedema, and certain electrolyte imbalances.

Cardiac irregularities

The second group of cases in which electrocardiography can be valuable is that in which irregularities or disturbances in mechanism are present. The more common irregularities are usually recognized at the bedside, but even these may prove difficult for most physicians. Others can be diagnosed in no other way than by a tracing. Electrocardiography is the final authority in irregularities unraveling the rhythms of the heart, and it can be considered the "living autopsy."

The clinician must realize that the correct interpretation of an arrhythmia may be the first clue to a previously unrecognized diagnosis. Furthermore, such a disturbance in the mechanism of the heart may endanger the life of a patient and may yet be curable.

When ordinary occasional extrasystoles are detected, it is rarely necessary to determine whether they are auricular or ventricular. There is too little difference in the clinical significance of the two types to warrant taking a tracing. In a rare case, however, it would help to determine whether attacks of paroxysmal heart action occurring in the same patient at other times were auricular or ventricular in origin. Inasmuch as such attacks may come and go so abruptly that it is almost impossible to obtain tracings of the episode itself, the findings of auricular extrasystoles during the free intervals would point more to paroxysms from the auricles, and the findings of ventricular extrasystoles would point more to paroxysms from the ventricles.

A gross irregularity is fairly typical of auricular fibrillation. A very rapid beat that comes and goes suddenly is characteristic of paroxysmal auricular tachycardia. When the rapid rate is due to auricular flutter or ventricular tachycardia, the diagnosis is not so simple. Since therapy in the two conditions is so different, and since the proper therapy may mean the difference between improving and worsening the situation, it follows that whenever an unusually rapid heart action that is not readily identified is found, a tracing should be taken if possible. The important practical point is that paroxysmal ventricular tachycardia cannot be helped by digitalis and, in fact, is made worse by it. It usually, but not always responds to quinidine.

Other types of attacks in which an electrocardiogram is important occur. Patients may have faint feelings and even attacks of unconsciousness, and on later examination, show no abnormalities. A sudden marked slowing of the heart, such as occurs in complete heart block, or the sudden onset of a very rapid rate may result in temporary unconsciousness. What is not generally appreciated is that tracings between attacks may be normal. Even hearts subject to Adams-Stokes attacks will occasionally show no disturbance of the auriculoventricular conduction between attacks. It is necessary, therefore, to make every effort to study any disturbance of the heart during any such attacks.

Rheumatic fever

The third group of conditions in which electrocardiography is helpful is in certain cases of fever of undetermined origin. It is obvious that one does not need an electrocardiogram in every febrile case he sees. It a patient has pneumonia or typhoid fever or tuberculosis, a tracing would show nothing unusual, or if it did, it would offer nothing to alter the diagnosis, prognosis, or treatment. On the other hand, if the diagnosis is obscure, the electrocardiogram may furnish evidence that would aid in identifying the case as one of *atypical* rheumatic fever.

Rheumatic fever may run a peculiar course without arthritis, and the choreic form may be manifested as only a slight irritability. There may be no symptoms at all except a slight or marked involvement of the heart, and the condition may resemble tuberculosis because of weight loss, fatigue, and a slight fever.

It is in these *atypical* cases that an electrocardiogram may help in the diagnosis. The characteristic changes are (1) that seen in first degree heart block—that is, a prolongation of the P-R interval beyond the normal of 0.2 seconds in adults and 0.16 seconds in children; and (2) alteration of the S-T segment. If digitalis has not been given recently and coronary artery disease can be ruled out (and if the condition is not congenital, as rarely occurs), the assumption that the changes represent rheumatic fever is fairly safe. The only other common disease that produces this picture is diphtheria, and clinical confusion here is unlikely. There have been reported recently also cases of acute

gonococcal arthritis accompanied by myocardial involvement and heart block.

Misuse of Electrocardiography

A word of caution must be said against the *overenthusiastic use* of the electrocardiograph. The machine itself is fairly inexpensive and so simple to operate that there are thousands of them in use. Many inadequately trained individuals are assuming the responsibility of interpreting the tracings. Some of the most serious errors are made in trying to read too much into the tracing. Slight flattening of the T waves and notching of the QRS complex may be abnormal and yet lack clinical importance. We have become so conscious of coronary artery disease that there is a tendency to read into a case evidence of coronary sclerosis which may not be justified; and this error is often aggravated by similar misreading of the electrocardiogram.

Another main misuse of electrocardiography is in *prognosis*. There is no doubt as to the diagnostic value of the technique. The point is that, once the diagnosis is established, changes in the electrocardiogram offer little to the prognosis. Experienced clinical judgment may be far superior to the opinions of the so-called cardiographic expert. There is no great value in taking frequent tracings in a known case of coronary occlusion, unless there is some reason to think that some extension has taken place. Even then the clinical course is more likely to be the guide in prognosis and management. The abnormal electrocardiogram is less important than the circumstances under which it is taken. Also, the finding of an abnormal electrocardiogram does not mean that the patient's heart is necessarily the cause of the patient's primary complaint.

Summary

There are three types of cases in which the general practitioner may find electrocardiography helpful: (1) any type of acute or chronic coronary artery disease; (2) arrythmias which may be difficult or impossible to diagnose; (3) atypical cases of acute rheumatic fever.

There are some cases that can be diagnosed by electrocardiography that cannot possibly be appraised by any other means. The diagnostic value may be great, but its prognostic value is limited. In prognosis, clinical evaluation is superior to statistical averages in relation to electrocardiographic changes.

Serious errors may be made by attaching too much clinical significance to minor abnormalities. The electrocardiographic report is only as good as the judgment of the interpreter. A normal electrocardiogram does not guarantee a normal heart or eliminate the possibility of organic heart disease. It does aid in the management of some patients who would not otherwise survive.

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RECURRENT PNEUMOTHORAX ASSOCIATED WITH HEREDITARY DEFORMING CHONDRODYSPLASIA

Report of a Case Apparently Due to Puncture of the Lung by an Exostosis of a Rib

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Hereditary deforming chondrodysplasia is an unusual condition which may affect all bones except those of membranous origin[1]. The ribs are involved in some cases and in others occasional pleurisy or pain in the breast has been reported, but no cases associated with pneumothorax have been encountered in a review of the literature[2]. In the case to be reported, observations made at operation seemed to confirm the clinical impression that the calcified exostosis had punctured the lung and had thus played a

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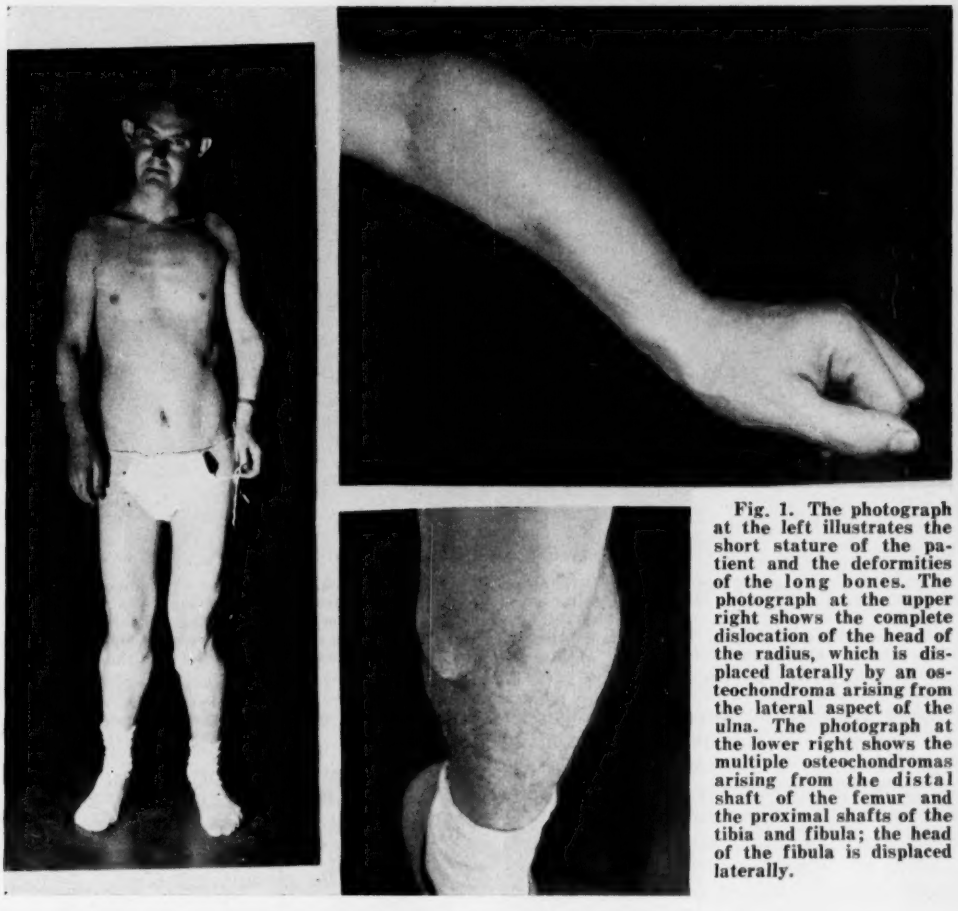

Fig. 1. The photograph at the left illustrates the short stature of the patient and the deformities of the long bones. The photograph at the upper right shows the complete dislocation of the head of the radius, which is displaced laterally by an osteochondroma arising from the lateral aspect of the ulna. The photograph at the lower right shows the multiple osteochondromas arising from the distal shaft of the femur and the proximal shafts of the tibia and fibula; the head of the fibula is displaced laterally.

part in producing the recurrent pneumothorax.

Case History

A 27 year old white male druggist was admitted to the North Carolina Baptist Hospital on August 20, 1948, complaining of dyspnea and pain in the chest.

Since early childhood he had been subject to the development of bony outgrowths, especially near the epiphyses of the long bones. The patient believed that these usually followed local trauma. When he was 18 years of age, one of these outgrowths was removed from the lower end of the right femur. The pathologic examination revealed several irregular fragments of cancellous bone covered by hyaline cartilage; the bone marrow was fatty.

Three months prior to admission he had experienced sudden severe chest pain in the region of the left nipple after sneezing. The pain was followed within a few minutes by marked dyspnea. Roentgen films of the chest showed a pneumothorax on the left side. The patient remained in bed for three days, during which time the lung began to re-expand. He then resumed light work. Three weeks before admission he had a similar attack while walking, but this time the dyspnea persisted.

The patient's mother and maternal grandmother also had bony outgrowths. A review of his past history did not reveal any serious illnesses.

On physical examination the temperature was 99.2 F, the pulse 96, respiration 18, blood

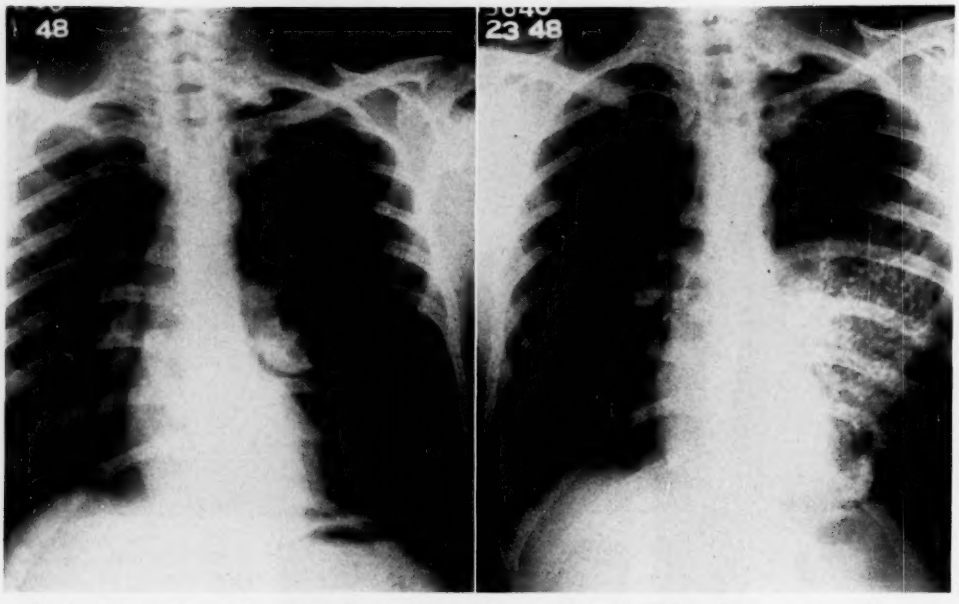

Fig. 2. The roentgenogram at the left, taken on the day after admission, shows a pneumothorax in the left pleural cavity with 60 per cent collapse of the lung. An osteochondroma arising from the eighth rib at the left lateral chest wall just above the diaphragm can be seen projecting into the left pleural cavity. An enlarged view of this lesion is seen in figure 4. The film on the right, taken two days later following the instillation of iodized oil at bronchoscopy, shows no local lesions in the bronchus or lung.

pressure 115 systolic, 90 diastolic. The patient was short of stature, and showed obvious deformity of the long bones (fig. 1). Numerous hard, non-tender bony projections were readily visible around the elbows, wrists, and knees. The upper end of the left ulna was broadened, as were the upper ends of both tibias. Both arms were short, the radiuses being gently curved. The right arm and leg were shorter than the left. A bony prominence was palpable over the fourth costal cartilage on the left.

Over the left lung the percussion note was hyperresonant, breath sounds were absent, and voice sounds were diminished. The mediastinum was shifted to the right, the cardiac apex being felt 1 cm. to the left of the sternum.

A hairy, elevated, brown-pigmented nevus was seen on the skin of the right forearm. This had been present since birth.

Blood counts and urinalysis were normal. The sedimentation rate was 3 mm. per hour. A Kahn test on the blood was negative. Smears and cultures of the sputum were negative for acid-fast organisms and for fungi. Intradermal skin tests with tuberculin, histoplasmin, and coccidioidin were negative.

The serum calcium was 10.3 mg. per 100 cc., the serum phosphorus 3.7 mg. per 100 cc., and the alkaline phosphatase 2.2 Bodansky units per 100 cc. The total serum proteins were 6.3 Gm. per 100 cc.—the albumin 4.0 Gm. and the globulin 2.3 Gm.

Roentgen examination of the chest showed a pneumothorax on the left side, with about 60 per cent collapse of the lung (fig. 2). Bronchograms outlined the collapsed lung, but demonstrated no parenchymatous disease. Multiple osteochondromas were seen arising from the ribs and from the transverse processes of the thoracic spine. Films of the left knee showed multiple large osteochondromas arising from the distal shaft of the femur and from the proximal shafts of the tibia and fibula. The head of the fibula was displaced laterally by a large osteochondroma. The distal end of the femur and the proximal portion of the tibia were considerably expanded, but the articular surfaces in the knee joints appeared normal. The left elbow showed marked deformity of both the radius and the ulna, with complete dislocation of the head of the radius. The radius was dis-

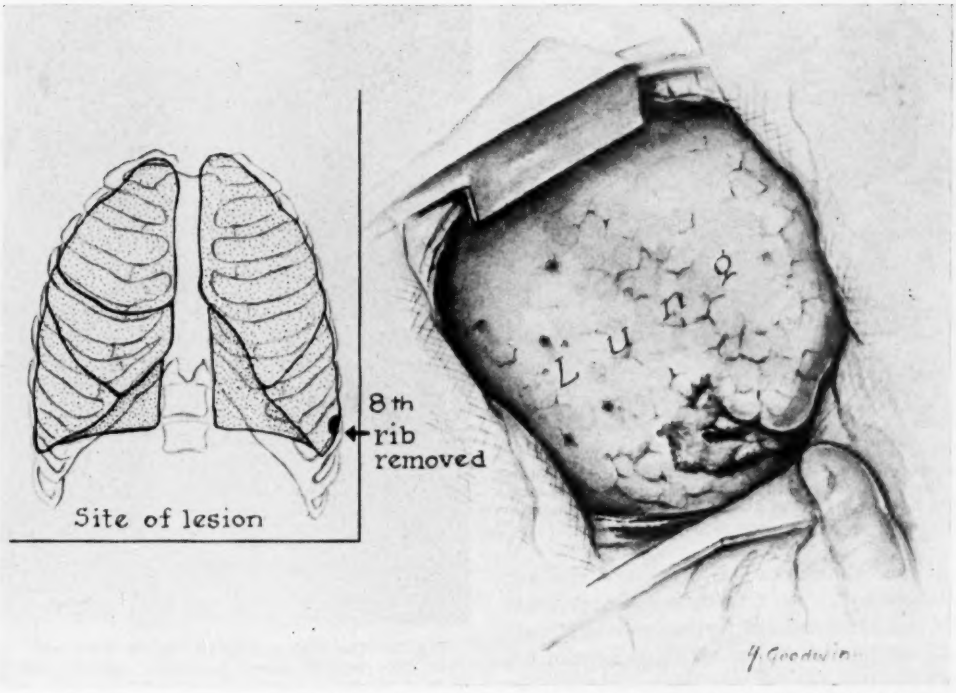

Fig. 3. The diagram on the left shows the location of the pointed exostosis on the eighth rib seen in the roentgenogram at the left of figure 2. The drawing on the right was made after the seventh rib had been removed and the lung expanded with positive pressure. The fibrosed collapsed area with an indentation suggesting a point of puncture is seen just above the retractor at the lower portion of the field.

placed laterally by a large osteochondroma arising from the later aspect of the ulna.

Following a negative bronchoscopic examination, an exploratory thoracotomy was performed. When the left pleura was opened, osteochondromas measuring from 2 to 5 cm. in diameter were found on the first, fourth, sixth, and eighth ribs. The costochondral junctions on the left side were all very prominent. On the eighth rib, near the costochondral junction, was seen a very prominent osteochondroma with a slightly rounded point of firm tissue projecting into the chest cavity. Opposite the point of the exostosis an imprint was clearly seen on the lower lobe of the left lung (fig. 3). When the lung was expanded by positive pressure, an area approximately 3 cm. in diameter, which appeared to have fibrosed as the result of repeated trauma, remained collapsed. No bronchopleural fistula could be demonstrated. A 7.5 cm. section of the eighth rib near its anterior border was removed (fig. 4). Sterile talc was rubbed over the visceral pleura to promote adhesions.

Pathologic examination of the operative

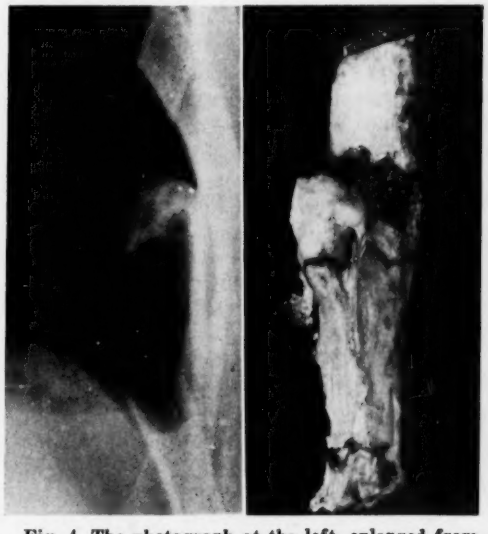

Fig. 4. The photograph at the left, enlarged from a roentgenogram of the chest, shows the pointed partially calcified osteochondroma arising from the eighth rib. The photograph at the right shows the 7.5 cm. portion of the rib including the osteochondroma removed at operation.

Fig. 5. The low power photomicrograph of a cross section of the osteochondroma illustrated in figure 4 shows the projection of old and new cartilage covered by dense fibrous tissue into the bone marrow.

specimen showed cartilaginous tissue surrounded by dense, fibrous connective tissue. Old and new cartilage formation was present, and some areas of new cartilage formed projections into the fibrous tissue (fig. 5).

The postoperative course was uneventful, and the patient was discharged on the tenth postoperative day with the lung 20 per cent collapsed. Later chest films showed complete re-expansion of the lung.

Five months after discharge—in February 12, 1949 — pain accompanied by dyspnea again developed in the region of the left nipple. Once again physical signs of pneumothorax were found. Roentgen examination of the chest showed 30 per cent collapse of the left lung; the base of the lung appeared to be adherent to the diaphram so that the lower lobe remained partially expanded (fig. 6). After rest in bed the lung re-expanded, and no further attack occurred for more than a year.

On April 15, 1950, while reaching upward toward a high shelf, the patient again experienced pain in the left side of the chest. Roentgen examination revealed a pneumothorax involving only the upper third of the left lung. No treatment was required.

When he was last seen—in August 1951—the patient was working full time, but had recently suffered another pneumothorax on the left. Roentgenograms revealed that the rib removed was regenerating.

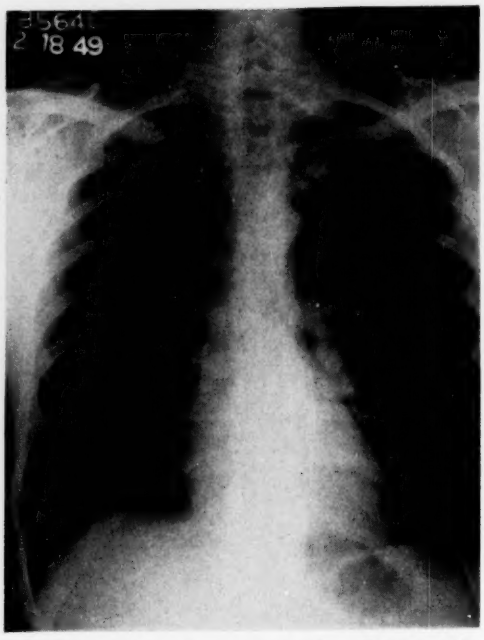

Fig. 6. The roentgenogram, taken February 18, 1949, five months after discharge, shows a small area of recurrent pneumothorax over the upper and lateral portions of the lung; the base of the lung is adherent to the diaphragm inferiorly and to the parietal pleura laterally. The left seventh rib was removed at the time of operation in order to enter the pleural cavity.

Comment

Hereditary deforming chondrodysplasia is a disease characterized by deformities of the long bones, with exostoses near the ends. Among the many synonyms which have been used to describe this condition are "multiple cartilaginous exostoses," "hereditary multiple exostoses," "multiple hereditary osteochondromatosis," diaphyseal aclasis," and "multiple congenital osteochondromata" [2,3,4,5]. The variable clinical picture of the disease probably accounts for the multiplicity of descriptive titles. The projections usually appear in childhood, grow most rapidly during adolescence, and tend to become calcified as the individual reaches maturity. The rib projections arise chiefly from the costochondral junctions; the skull and spine may also be affected.

The etiology has been widely discussed. The presence of bony deformities in this patient's maternal ancestors seems to leave no doubt that the present case was hereditary as well as congenital in origin.

The lesions probably begin as an overgrowth of cartilage cells from the epiphysial plate. Since the growth is unchecked by the periosteal sleeve, the cartilage grows outward as a projection and later becomes calcified; the epiphyses tend to become widened and deformed[2]. Attempts have been made to reproduce the lesions experimentally by fracturing the long bones and by grafting pieces of bladder mucosa into the rectus muscle of the dog[6].

Spontaneous pneumothorax is known to occur following pulmonary disease — most frequently tuberculosis, although malignant growths and other conditions have also been incriminated[7]. Following strain, pneumothorax may result from the rupture of an emphysematous bleb on the surface of the pleura or from the rupture of an adhesion[8]. Pneumothorax also occurs spontaneously in normal individuals without pulmonary disease in whom no cause for the pneumothorax can be found[9].

In the present case, no evidence suggesting an infectious etiology could be found. Strain was minimal, since the patient did not lift heavy weights or do hard work. In view of the upward angle of projection of the exostosis, the occurrence of pneumothorax after sneezing and after reaching would be compatible with the impression that the sharp cartilaginous tissue may have been responsible. The incomplete pneumothoraces which occurred postoperatively could have been produced by the rupture of small adhesions. Previous attempts to produce adhesions between the visceral and parietal pleuras by the instillation of talc have been reported[10]. In this case the attempt was partially successful, as the roentgenograms illustrate (fig. 6). The reduction in the extent of subsequent collapses indicates that some benefit was derived from the pleural reaction induced at operation.

Summary

A case of spontaneous pneumothorax occurring in association with hereditary deforming chondrodysplasia is presented. Since no bronchopleural fistula was demonstrated at operation, it is difficult to be certain that the osteocartilaginous projection from the eighth rib into the left pleural cavity actually punctured the lung and produced the pneumothorax. The imprint of the exostosis on the lung and the presence of scar tissue, however, suggest an etiologic relationship.

Removal of the involved portion of the rib and the production of adhesions between the parietal and visceral pleuras by the instillation of sterile talc has reduced the number and severity of the recurrent pneumothoraces.

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Shortage of x-ray films: The consumption of x-ray films in hospitals and clinics is difficult to limit, particularly in large departments which strive for a high standard. Wastage occurs each winter by a variation in power at peak hours which disorganizes the exposure charts of every radiographer. Films are wasted by the duplication of examinations in out-patients and wards, by requesting films of both knees when one is affected, by ordering before the former film has been inspected. These are obvious but common mistakes. Equally appreciated is the zealous houseman who tries to anticipate every wish of his consultant. The arrival of a new batch of medical officers is reflected in an increase in the work of auxiliary departments, but this increase is temporary. — Editorial, British Medical Journal 1:94 (Jan. 12) 1952.

Another interesting ebb and flow in medicine concerns itself with changes in the severity of illness caused by certain diseases—not related to improvements in methods of prevention or treatment or both. As an example, in my lifetime I have observed a striking and rather steady decrease in the severity of rheumatic fever, although there seems to have been no corresponding decrease in the seriousness or frequency of valvular heart disease of rheumatic origin. Likewise, I have twice seen influenza change from a rather mild illness to a serious one of pandemic proportions of high fatality. Bright's disease also has seemed to lessen in the severity of its manifestations, in a way not to be explained by any great improvement in its therapy and at a time when antecedent, probably causative bacterial infections were no better controlled or treated. This occurred in the period prior to the use of sulfonamides and antibiotics in these infections.—Christian, H. A.: Ebbs and Flows in Medical Interest, New England J. Med. 246:284 (Feb. 21) 1952.

SEVERE CHOREIC STATES IN ADULTS AS MANIFESTATIONS OF DEPRESSIVE REACTIONS

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and
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Little is known about the pathology or altered physiology which may be presumed to cause most mental illnesses. Likewise, little is known about what the physical therapies such as prefrontal lobotomy, electric shock treatments, insulin, and carbon dioxide accomplish physiologically or structurally to bring about clinical improvement or cure in the treatment of mental illnesses. Therefore, it seems worth while to add to the literature as many observations as possible concerning the relief of mental illnesses with physical therapies in the hope that some day the literature may be reviewed for the dual purpose of trying to discover the etiologic factors responsible for mental illnesses and the modus operandi of the several physical therapies.

This paper is a report upon the treatment of two severe choreic states in adults who were treated with electro-convulsive therapy. It is believed that each of these patients had a primary depressive mood disturbance and that the choreiform movements were secondary to the affective disorder.

Case 1

First hospital admission

The first patient, a 46 year old woman, was admitted to Duke Hospital on December 31, 1946, complaining of weakness of the right leg and arm and of involuntary jerking movements involving the right side of the face, the right arm, and the right leg of three months' duration. The onset of the jerking movements was preceded by pain in her right leg, which began in February, 1946. Following the onset of this pain, she had difficulty in sleeping, lost weight, refused to have sexual intercourse with her husband, and became extremely nervous. She took bromides as prescribed by her physician until she had a skin rash and became confused and somewhat paranoid. The patient remained at the hospital for 23 days, during which time she received psychotherapy. At the time of discharge her family thought she was improved, but the house officer who wrote the discharge note said that he could see no change in her condition.

The history revealed that the patient comes from a somewhat unstable, peculiar, and quarrelsome family. She was said to have been treated for a speech difficulty at about the age of 9. One sister was married to an alcoholic husband; and a brother had been treated for syphilis. This brother had sued the estate upon the death of the patient's father. The patient's mother had died of a right-sided paralysis. A further disturbing factor was the recent amputation of a neighbor's leg because of hardening of the arteries and gangrene of the leg.

The patient married at the age of 18. Two years later she gave birth to a daughter, her only child. Subsequently the patient had at least two miscarriages.

The patient had been hospitalized in 1938 at the age of 26 because of palpitation, perspiration, shaking, a lumpy feeling in her throat, weakness in her knees, and nervousness.

Two years prior to the onset of the present illness, her only child married, and the patient was left alone with her husband. He was frequently away on prolonged business trips, during which she frequently wrote and accused him of drinking and running around with other women.

Second hospital admission

The patient was re-admitted to Duke Hospital on October 30, 1950. At that time her condition was reported to have remained about the same except for increasing nervousness, loss of weight and appetite, and accentuation of the involuntary movements of the right side of her body during the past year. Six weeks prior to re-admission, rather pronounced vaginal bleeding had developed, and three weeks later a dilation and curettage was performed. Ten days prior to admission she began sticking out her tongue involuntarily, and hyper-extending her neck; and the involuntary movements, which previously had been present only on the right side of her body, became generalized. She had been taking bromides, and her blood bromide two days prior to admission had been reported to be 550 mg. per 100 cc.

At the time of this admission, the patient was emaciated and extremely agitated. She was flailing her arms and legs in aimless choreiform movements to such an extent that she had to be continually restrained from adding new bruises to her already badly bruised body. These movements could be markedly reduced by voluntary effort, especially if her attention could be distracted.

An electroencephalogram done under Sodium Amytal narcosis showed no focal abnormalities, and seizure discharges did not occur. Her blood bromide several days after admission had dropped to 24 mg. per 100 cc.

Ten days after admission the patient's involuntary activity continued to increase until it was thought that she would literally beat herself to death unless drastic measures were taken. She was given two electric shock treatments daily for five days, and one treatment a day for six additional days. All the treatments resulted in major convulsive seizures, although on the days when two treatments were given the second seizure was less severe than the first.

The patient did not begin to show definite evidence of post-shock confusion until after her fifteenth treatment. After the sixteenth, she was definitely confused, but her involuntary movements were significantly decreased. In the early morning hours of the night after her last treatment, the patient suddenly became extremely violent, screamed, yelled, and attacked her private duty nurse. It required the concerted efforts of an orderly, an interne, and two nurses to restrain her. She was transferred to the disturbed psychiatric ward, where she continued to be disturbed and confused for four days.

On the fifth day her mental condition rather

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From the Department of Psychiatry, Duke University School of Medicine, Durham.

suddenly cleared. She dressed herself, applied make-up, and was permitted to go to the day room, where she behaved quite normally. Three days later her behavior was still normal, and she was permitted to go home. One year later a letter written by the patient with pen and ink and in excellent penmanship was received. She reported that she had been completely well since leaving the hospital.

Case 2

First hospital admission

The second patient was a 52 year old, married, white woman who came to the hospital on November 14, 1951, with a chief complaint of intermittent pain in the right foot just distal to the heads of the third and fourth metatarsals of 10 or 15 years' duration. She stated that these attacks of pain were precipitated by being on her feet for more than three hours, and had been more frequent and severe during the past six months. In addition to the pain in her right foot, she also had pain in both thighs and legs.

The patient's hospital record showed that she had been seen on April 16, 1937, with the complaint of severe pain in her left jaw, which made chewing or talking almost impossible. She had had this pain for two and one-half months. The orthopedic surgeon who saw her at that time thought that she had synovitis or arthritis of the left maxillomandibular joint. Her symptoms had been so severe that the orthopedist considered having her teeth wired together in order to give her jaw complete rest. However, the condition subsided promptly when the patient was put on a liquid diet and told not to move her jaw. The hospital record further revealed that from 1943 to 1948 she had received symptomatic treatment for what was thought to be sciatica on the right.

She was seen in consultation by the orthopedic service, and a diagnosis of plantar neuroma of the right foot was made. An operation lasting one hour was performed under Pentothal Sodium anesthesia on December 4, 1951. The growth was removed and the diagnosis confirmed by histologic examination of the tissue. At the completion of the operative procedure, apnea developed in the patient and lasted five minutes. Artificial respiration was administered, and the patient did not become cyanotic.

At 6:50 p.m. on the day of operation, rather violent movements of her trunk and extremities began, with simultaneous panting and groaning. Some observers thought her behavior resembled that of a woman about to experience orgasm during sexual intercourse. She was conscious, oriented, and able to carry on a conversation intelligently, but stated that she was unable to stop these movements. There were no localizing neurologic signs; 8 cc. of chloral hydrate given by mouth made the patient drowsy but did not alter her behavior. One and one-half hours after the onset of her involuntary activity, she was given 100 mg. of Demerol intravenously by the anesthetist, and she went to sleep and remained quiet throughout the night. She had no further involuntary movements throughout her stay in the hospital. The next morning on being questioned about her experiences of the day before, she stated that she could remember only that she had a nightmare.

The patient remained in the hospital for one week following her operation. During this time she complained of weakness and did not want to get out of bed when advised that she could. She seemed to be somewhat in a daze, picking up a newspaper and laying it aside with the remark that she could not concentrate.

Second hospital admission

Three days after leaving the hospital (10 days after operation), the patient became nervous and restless. That night she did not sleep well. The following day the involuntary writhing movements were resumed and continued all night, keeping both the patient and her husband awake.

Twelve days after the operation, she was readmitted to the hospital. At this time the violence of her movements was causing her to bruise herself in many places. One hundred mg. of Demerol given intravenously quieted her only temporarily. She was first seen by us the next day, when two nurses were required to prevent her from self-injury. The patient was interviewed under Pentothal narcosis, but no significant information could be obtained. It was suggested to the patient that her movements would quiet down when she awakened, but they did not.

The next morning, during a second interview conducted under Sodium Pentothal, the patient was questioned about her sex life. She stated that she had had no sexual desire since she went through the menopause four years ago, that she had not had sexual relations for four or five years, and that her husband was impotent. Her movements seemed to be greatly intensified while we were talking about sexual matters. During this second interview she also reported that she had been depressed ever since her mother died 10 months ago. Upon awakening from this second interview, her movements were just as violent as before.

The patient was then given three carbon dioxide treatments, without effect. She was then started on electro-convulsive therapy and her involuntary activity immediately began to subside. After four treatments her movements had almost stopped. At this time the patient showed definite evidence of depression, and had several crying spells each day. She was given three additional electric shock treatments for a total of seven treatments during a period of five days, and was permitted to leave the hospital with instructions to report back for outpatient observation and treatment as necessary.

Third hospital admission

The patient did quite well from the time that she received her last electric shock treatment on December 23, 1951, until February 2, 1952, at which time she reported back to the hospital complaining of mild depression and of a recurrence of involuntary movements in the fingers of her left hand and in the muscles of her left lower extremities. She was given another electric shock treatment on that date, and these movements subsided considerably. She was given five more electric shock treatments for a total of 14, but the only observable result was the development of confusion. She still had some difficulty in manipulating her silverware while eating because of the tendency toward involuntary movements of the fingers of the left hand, and there was some difficulty in walking because of involuntary muscle spasms in the muscles of her left lower extremities. At the present time the patient has only occasional choreiform movements in her left hand and is doing quite well.

Comment

When these two cases are compared, they are seen to have certain characteristics in common:

1. The adventitious movements were either unilateral in the beginning and then became bilateral, or were bilateral in the be-

ginning and then became unilateral during the course of treatment.

2. Each of the two patients gave a history of long-standing instability with a tendency towards hysterical-like, somatic complaints.

3. Each of the patients gave a history of disturbed sexual activity.

4. The two patients were not relieved of their adventitious movements until shock therapy had produced signs of post-shock confusion.

Conclusions

It is concluded that these two patients were relieved of their choreiform movements by electric shock treatment because their choreiform movements were somatic manifestations of a depressive affective disorder. Since rather intensive treatment was required and since the choreiform movements did not cease until the patients showed definite memory disturbances and confusion, we are led to believe that the movements stopped as a result of the depressing effect of the electric shock upon the functioning of the neurons in the cerebral cortex.

It seems worth while to contrast this hypothesis with the hypothesis that stimulating nonconvulsive electric shock treatments relieve psychic pain by their stimulation of subcortical structures. It has been our experience that patients who are relieved of psychic pain by electric shock show evidence of rather marked sympathetic nervous system responses to the treatments, without manifestating any signs of confusion or memory disturbances.

Discussion

Dr. Leslie B. Hohman (Durham): I think these 2 cases illustrate part of a thesis which we uphold—namely, that a great many of the so-called psychoneuroses are really affective depressions and that the manifestations of hysteria, anxiety, or neurasthenia are symptomatic of affective disorders. We believe that the proof of this theory lies in the recoveries that are achieved, since it is generally agreed that electric shock is valueless except in the affective disorders.

The Effect of the Soma on the Psyche: Awkwardness and inexperience in the handling of patients in the early stage of their illness may lead to serious trouble, in the form of psychoneurosis, depression, lack of cooperation in the essential treatment, and distrust of the medical profession. The cults, including Christian Science, have in large measure thrived as the result of our ineptitude in this particular.—White, P. D.: The Psyche and the Soma, Ann. Int. Med. 35:1297 (Dec.) 1951.

APLASTIC ANEMIA WITH QUESTIONABLE RELATION TO CHLOROMYCETIN THERAPY

Report of Two Cases

J. L. COCHRAN, JR., M.D.

CHARLOTTE

Bone marrow depression has been observed during short periods of Chloromycetin therapy[1]. Only 1 case of fatal aplastic anemia developing during prolonged use of Chloromycetin therapy has been reported in the English literature[2]. The following 2 cases of fatal aplastic anemia are reported because of their possible relationship to Chloromycetin therapy.

Case 1

A white male, age 4 years, was in excellent health up until July 8, 1951, when malaise, anorexia, headache, and a fever of 103 F. developed. Physical examination did not reveal the cause of the illness, but some redness of one ear drum was noted. The patient was started on Chloromycetin, 250 mg., four times a day, and received a total of 18 doses. The only other medication given was aspirin, 2½ grains, on one or two occasions. The patient's fever subsided, and he appeared well. However, the mother felt that he was not as active and full of life as usual, and he continued to show anorexia and easy fatigue. On August 15, 1951, the mother noted over the patient's body large ecchymotic areas which were thought to be traumatic. Two days later the patient was bleeding from the gums and was hospitalized.

Past history

The past history revealed that the patient had always been in excellent health. He had never received medications except small amounts of aspirin on rare occasions. The history for exposure to drugs, chemicals, plant sprays, and so forth, was completely negative.

Physical examination

The patient was a well developed child and did not appear acutely ill. The rectal temperature was normal. Positive physical findings were limited to the skin and mucous membranes. There was a large ecchymotic area over the left iliac crest, and several smaller areas over the lower extremities. A few petechial lesions were noted in the mouth and over the trunk. There was no lymphadenopathy or jaundice. The liver and spleen were not palpable. A tourniquet test was markedly positive.

Laboratory findings

The urine was negative on routine examination. The red blood cell count was 3.18 million; hemoglobin, 9.0 Gm.; white blood cell count, 9,000; polymorphonuclears, 27 per cent; lymphocytes, 63 per cent; eosinophils, 10 per cent. The platelet count was 125,000. A platelet count four days later showed 60,000. Bleeding time was 2.5 minutes; clotting time, 4 minutes; clot retraction, slight in 24 hours. A stool examination was negative for ova and para-

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Read before the Section on Pediatrics, Medical Society of the State of North Carolina, Pinehurst, May 7, 1952.

From the Department of Pediatrics, Charlotte Memorial Hospital, Charlotte, North Carolina.

sites. Bone marrow aspiration on two occasions showed a marked hypoplasia of all bone marrow elements.

Clinical course

The patient's white cell count slowly decreased to around 3,000, with 100 per cent lymphocytes. The last platelet count taken was 14,000. Bleeding from the nose, gums, and gastrointestinal tract occurred at intervals, and was controlled with transfusions of fresh blood. The patient was given liver by injection and vitamins as supportive measures.

In November, 1951, while at home, the patient developed a nasal discharge of clear mucus. This nasal infection rapidly progressed into ethmoiditis and cellulitis of the right side of the face. The patient was admitted and treated vigorously with surgical drainage, antibiotics, and chemotherapy. Culture of the nasal discharge showed hemolytic **Staphylococcus aureus** and beta streptococcus highly resistant to all drugs tested. The infection progressed into gangrene of the face, nose, and palate. Signs of generalized septicemia developed, and the patient expired on December 6, 1951.

Autopsy findings

Bone marrow from rib, sternum, and vertebrae showed marked hypoplasia involving the granulocytic and erythroid series. There were numerous iron-containing macrophages, innumerable plasma cells, as well as multinucleated and giant cells taken to be proliferating reticulum cells.

All major organs showed numerous bacterial colonies and Monilia colonies. The stomach and intestinal tract up to the rectum showed innumerable ulcerations associated with the growth of Monilia colonies.

Case 2

A 6 year old white boy had been well up until July 14, 1951, when he contracted an illness characterized by headache, nausea, vomiting, and fever ranging from 100 to 103 F. The cause of the illness was not apparent on physical examination. The patient was started on Chloromycetin, 250 mg., four times a day. He received the drug for four days. By this time, the patient's temperature was normal and he appeared well. Approximately one week later the symptoms returned, and the patient was given Chloromycetin, 250 mg., four times a day for three days. The patient appeared to recover. This was around July 28, 1951. During the month of August the patient slowly developed pallor, exertional dyspnea, and preferred to sleep most of the time. A few areas of ecchymosis were noted at intervals, but were thought to be traumatic. The areas of hemorrhage began to appear in alarming numbers, and the patient was hospitalized on September 11, 1951.

Past history

The past history revealed that the patient had been in good health and had never been seriously ill. Since two years of age, he had had occasional attacks of hay fever treated with Benadryl. He had not had an attack for approximately eight months.

There was a negative history for exposure to poisons, paints, or other medications.

Physical examination

The patient was a well developed white male. The temperature was normal. The mucous membranes, skin, and bony prominences showed numerous petechial and ecchymotic lesions. There was no jaundice or lymphadenopathy. The liver and spleen were not palpable. A tourniquet test was markedly positive. The remainder of the physical examination was not remarkable.

Laboratory findings

The urine was negative on routine examination. The red blood cell count was 1.8 million; hemoglobin, 5.0 Gm.; white blood cell count, 5,150; polymorphonuclears, 5 per cent; lymphocytes, 96 per cent. The platelet count was 40,000; clotting time, 3 minutes and 11 seconds; bleeding time, 18 minutes; clot retraction, none in 18 hours. A histologic examination of bone marrow showed a marked diminution in myeloid and erythroid elements, with only infrequent marrow elements scattered within fat tissue.

Clinical course

The patient was admitted at intervals and given transfusions of fresh blood for the control of bleeding from the gums, nose, and gastrointestinal and urinary tracts. In addition, he was given vitamins, oral liver concentrate, and a penicillin tablet (100,000 units) three times daily. On one occasion there developed on the leg a large abscess which responded well to terramycin. Infection was never a particular problem.

The patient's white blood cell count ranged around 2,000, with a differential count of 3 to 8 per cent polymorphonuclears, the remainder being lymphocytes. The platelet count varied around 8,000 to 10,000. Bleeding gradually became more pronounced, and frequent transfusions were necessary.

On October 16, 1951, the patient was started on Cortone, 25 mg. twice daily. Approximately six weeks later he began to complain of pain in the thighs, which he described as "nervousness." He insisted on rocking to and fro in a knee-chest position, and was noted to have an erection much of the time. The patient traumatized his face with this activity and produced conjunctival hemorrhage and bleeding from the nose and lips. On January 4, 1952, after increasing amounts of sedatives failed to control the patient, the Cortone was stopped. Within 36 hours, the patient became quiet and complained no longer of pain.

On January 15, 1952, following a cerebral hemorrhage, he expired.

Autopsy findings

Innumerable discrete and confluent areas of recent hemorrhage were found in the brain, subarachnoid and subdural spaces, and in the kidneys, urinary bladder, gastrointestinal tract, skin, lungs, striated muscle, and the heart. There was massive retroperitoneal hemorrhage involving both the anterior and posterior abdominal walls and pelvis. The spleen weighed 78 Gm. and showed a normal architectural pattern. The adrenal glands appeared normal. The bone marrow in the ribs, vertebrae, and sternum was reddish-brown in color and slightly firmer than usual. Microscopically, there was a marked absence of hemopoietic tissue. Only spotted areas of hemopoiesis were found. No other abnormalities of the hemopoietic system were demonstrated.

Summary and Conclusion

Two cases of fatal aplastic anemia following Chloromycetin therapy are reported. In each case, Chloromycetin therapy had terminated four to five weeks before distressing clinical symptoms brought the patient to a doctor.

The evidence as to the causative agent in each case is circumstantial. However, marked bone marrow depression during short periods of Chloromycetin therapy have been pre-

viously observed and reported in the literature. In the cases reported by Volini[1b], the peripheral blood count returned to normal following withdrawal of the drug. It is believed that Chloromycetin produced irreversible changes in the 2 cases reported here.

The second patient was given Cortone without detectable effect on the clinical course or peripheral blood picture. The only disturbing side effect noted was interpreted as an increase in the sexual drive of the patient.

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AN ANALYSIS OF MATERNAL MORTALITY IN CHARLOTTE AND MECKLENBURG COUNTY, 1938-1951

O. HUNTER JONES, M.D.

CHARLOTTE

The Metropolitan Life Insurance Company states that today the hazards incidental to pregnancy and childbirth are a level which seemed Utopian not many years ago. Currently the rate is appreciably under 1.0 per 1,000 live births nationally, and in large sections of the country it is even less than 0.5 per 1,000 live births. The rate has been reduced by about two-thirds in the past five years. In North Carolina in 1914 the maternal rate was 7.3 per 1,000 live births, while in 1951 it was 1.0 per 1,000 live births.

Significant Facts

1. There has been an amazing drop from 5.7 per 1,000 live births in 1938 in Charlotte and Mecklenburg County (considered together) to 0.3 per 1,000 live births in 1951, a period of 14 years.

2. The rate for Negro patients has been consistently higher than that for white patients, with the exception of the year 1939.

3. There were no deaths among white patients (out of a total of 8,970 live births) in 1949 and 1951, and only 1 death out of a total of 13,174 live births in the three year span of 1949, 1950, and 1951. In the same period there were 6 deaths among Negro patients in a total of 4,329 live births. If the year 1948 is included, however, 5 additional deaths among white patients occurred, or for the four year period of 1948-1951, 6 deaths in 17,605 live births. Likewise, 4 additional deaths occurred among Negroes, or for the four year period of 1948-1951, 10 deaths in 5,538 live births.

Table 1
Maternal Mortality in Mecklenburg County Compared With State and National Maternal Mortality 1938-1951
(Rate Per 1,000 Live Births)

Year	Mecklenburg County			N.C.	U.S.
	White	*Negro*	*Total*		
1938	5.5	6.1	5.7	5.6	4.4
1939	5.3	4.6	5.0	5.0	4.0
1940	4.1	8.7	5.3	5.2	3.8
1941	4.9	6.4	5.3	4.0	3.2
1942	2.7	8.2	4.2	3.5	2.6
1943	2.3	4.9	3.0	3.3	2.5
1944	1.3	4.0	2.0	3.0	2.3
1945	2.0	5.0	2.8	2.7	2.1
1946	0.3	4.9	1.3	2.1	1.6
1947	0.9	1.6	1.0	1.8	1.3
1948	1.1	3.3	1.6	1.9	1.2
1949	0.0	0.7	0.2	1.3	0.8
1950	0.2	2.8	0.7	1.2	0.7
1951	0.0	1.2	0.3	1.0	

Total live births 59,192; total deaths 128.

4. The Charlotte-Mecklenburg mortality rate has been consistently lower than the North Carolina state rate since 1946. Likewise, it has been lower than the national rate since 1946, except for the year 1948. At last North Carolina's maternal mortality rate is approaching a respectable figure, but there is still room for improvement. The Charlotte-Mecklenburg rate, if it continues to follow the trend of the past three years, is approaching the irreducible minimum—at least for white patients.

Is there an irreducible minimum? Dr. George Gordon Douglas, professor of obstetrics and gynecology at Cornell University, states in regard to Cornell's (the New York Hospital) rate: " It is apparent that obstetrical patients can now be cared for without mortality resulting primarily from the process of pregnancy or parturition, except in the rarest circumstances. There may continue to be occasional fatal accidents, but what was considered an irreducible minimum in this respect a decade ago has been reduced materially today . . . Survival alone should not be our only criterion of end results achieved."

Of the 128 deaths, 115 occurred in hospitals and 13 in homes. One death occurred with a midwife in attendance (home delivery).

In 1951, out of 6,183 live births, 207 infants, all Negro (135 city, 72 county) were

Table 2
Analysis of 128 Maternal Deaths in Mecklenburg County 1938-1951

Causes of Death			No. Deaths	Per Cent
Hemorrhage			31	24
A. As a direct cause of death				
Ectopic pregnancy	5			
Abortion	3			
Placenta previa	7	25		
Accidental hemorrhage	2			
Postpartum hemorrhage	8			
B. As an associated factor				
Exhaustion state, shock	4			
Rupture of the uterus	1	6		
Abdominal pregnancy	1			
Toxemia of pregnancy			43	34
Hyperemesis gravidarum	4			
Polyneuritis of pregnancy	1			
Nephritis and other renal lesions	15			
Eclampsia	23			
Infection			27	21
Abortion	14			
Puerperal sepsis	13			
Embolism (including 2 cases of amniotic fluid embolism)			9	7
Others			18	14
Pyelonephritis	3			
Cardiac failure	4			
Pernicious anemia, pneumonia, intestinal obstruction (2 cases), thrombocytopenic purpura, urinary suppression, colitis	8			
Undetermined	3			

delivered by midwives at home. In addition, 84 (including 23 white babies) were delivered by doctors in the home (39 city, 45 county).

Causes of Death

Toxemia of pregnancy leads the list in Charlotte as the cause of maternal death. In most other studies, hemorrhage leads the list—probably the greater availability of blood (blood bank) is a major factor in the reduced mortality from hemorrhage here. There has been only 1 death from puerperal sepsis in Charlotte during the past eight years. Two of the deaths from embolism were caused by amniotic fluid embolism. None of the remaining 7 deaths attributed to embolism were followed by autopsy, so it is possible that the diagnosis was incorrect in some of these cases. No deaths were attributed to anesthesia; however, in 1 case it was listed as a contributing factor. Eighteen autopsies were performed, giving a rate of 14 per cent; the rate for the past four years, however, has been 38 per cent.

In the past four years (1948-1951), there have been 16 deaths—5 from toxemia, 5 from hemorrhage, 2 from infection, 2 from amniotic fluid embolism, 1 from exhaustion due to a postpartum psychosis, and 1 associated with post-cesarean colitis (cause of death not fully determined).

As a basis for comparison, the maternal mortality rate in New York City was 1.1 per 1,000 live births in 1948 and 0.9 per 1,000 live births in 1949.

Conclusion

The reduction in mortality is a part of the country wide decrease. It represents better obstetrics, which means a higher standard of obstetric practice on the part of the physician; more hospital deliveries and better hospital facilities; higher hospital staff standards and requirements; more outpatient clinics; as well as education of the public and the desire for better care on the part of the patient. Certainly the availability of chemotherapeutic and antibiotic drugs, plasma, and blood has been a major factor. Much credit should also be given the State Maternal Welfare Committee. Finally, prosperity has undoubtedly been a factor.

This analysis was compiled solely from the death certificates filed with the local Health Department and tabulated by Mrs. Louise Young Workman, statistician.

Today, life expectancy is about 20 years longer than it was in 1900. More Americans can expect to live well into their seventies, eighties, and nineties. Aging brings with it new and challenging problems that must be recognized.

In an effort to help people understand what some of the common problems of aging mean to the individual, his family and his community, the Federal Security Agency has issued a guidance booklet entitled **Looking Forward to the Later Years.**

Copies of the publication (PHS Publication No. 116) can be purchased for 5 cents each from Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C. A discount of 25 per cent is allowed on orders of 100 or more sent to one address.

Ebbs and Flows in Medical Interest: As one grows old in medicine, he recalls ebbs and flows in the tides of medical interest in various diseases, as evidenced by periods when many articles are published on some specific subject and other periods when interest in that subject seems to wane for months or even years. In the course of time one can even observe an irregular periodicity of these cycles and mentally plot curves of ebb and flow. The full cycle is occasionally as short as five years; occasionally it lasts ten or fifteen years, or even longer.—Christian, H. A.: Ebbs and Flows in Medical Interest, New England J. Med. 246:281 (Feb. 21) 1952.

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"The prime object of the medical profession is to render service to humanity; reward or financial gain is a subordinate consideration. Whoever chooses this profession assumes the obligation to conduct himself in accord with its ideals."—Principles of Medical Ethics of the American Medical Association, Chapter 1, Section 1.

DECEMBER, 1952

NAVY SURGEON GENERAL MALIGNS CIVILIAN PHYSICIANS AND DENTISTS

In a prepared address delivered on November 17 before the annual meeting of the Association of Military Surgeons, Rear Admiral Lamont Pugh, Surgeon General of the Navy, asked the rhetorical question, "What is the main objection to the military and the main attraction to civilian medicine?" His insulting answer was: "We all know it is simply a matter of money-avarice, a better if albeit a fanciful and possibly an ephemeral opportunity to get rich quick."

One wonders just what moved Admiral Lamont to make such a malicious attack upon civilian physicians and dentists. Certainly he was mistaken if he thought it would encourage them to volunteer for the armed services. It is not the nature of doctors to be driven by browbeating and abuse. Had Admiral Pugh ever been a civilian doctor, he would have known this; but more than 25 years spent as a naval medical officer have apparently given him the rigid, domineering mind that is only too often acquired by men whose lives have long been regimented. The prospect of having such a man as "chief" is not likely to make physicians and dentists flock to his banners.

Had Admiral Pugh ever done a few years of honest, hard work as a civilian doctor—whether in general practice or in a specialty—he would have known that it is not an easy way to make money. Compared with the average general practitioner, the career men in the armed forces spend most of their lives in ease and comfort. Statistics have shown that the incomes of physicians have not risen in proportion to the cost of living generally, and the money earned by the average practitioner is neither quick nor easy.

Admiral Pugh further insults his own chosen profession when he wonders "if the doctor isn't on his way to becoming an heretic of the doctrine that it is more blessed to give than to receive." Can he name any member of society who gives more freely of his time, his energy, his hours of sleep and recreation, and his very self than does the average family doctor? It is unfortunately true that there are a few medical men who prostitute their professional attainments—but these constitute a small minority of our profession. Furthermore, in virtually every state medical society of the Union, grievance committees have been set up to discipline just such members.

Doctors have not forgotten, if Admiral Pugh has, the woeful waste of medical manpower in World War II. Letters from scores of medical men of all ages who were in service came to this office, complaining of the dreadful boredom from which they suffered with so little to do. The Duke Hospital Unit, "after five weeks of basic training and indoctrination in military procedures," was kept at Fort Bragg for more than a year before it was sent overseas. Even granting that "they also serve who only stand and wait," a year is a long time for a doctor to be professionally idle—especially when the proportion of doctors available for the civilian population was at a dangerously low level.

Admiral Pugh to the contrary notwithstanding, doctors would much rather *give* freely of their medical talents in order to keep them bright and shining than to *receive* more leisure than they want. Doctors are willing to serve their country when their country needs their services, but they want to guard against misuse of their professional training and skill. The fact that more than 60,000 physicians—one third of all the doctors in the United States—volunteered for service in the armed forces during World War II proves that they are not lacking in patriotism. Dentists also did not hesitate to volunteer when they were needed.

Admiral Pugh really outnodded Homer when he asked: "Has the doctor become a convert to a creed which during recent years has made great gains in popularity and in the number of its followers and whose motto is "Security above liberty?'" Certainly the members of no profession have fought harder than have the American doctors, both as individuals and in organizations, to combat the creed of "Security above liberty," which has been the theme song of the bureaucrats in Washington for the past twenty years. The recent election would indicate that the effort has not been in vain.

WHOSE BIRTHDAY DO WE CELEBRATE?

A recent *Saturday Evening Post* editorial tells of a Christmas party for Japanese Children given by a company of the U.S. Army of Occupation in Japan. As a G.I. in a red Santa Claus suit and cotton whiskers was giving out presents, 8 year old Ito asked a question that is hard to answer: "Whose birthday is it? The man with the whiskers' birthday?" Someone had told Ito that they were celebrating the birthday of the Saviour —but to him it seemed that Santa Claus was the main attraction.

It must be admitted that too many adult Americans are forgetting that the real purpose of Christmas is to celebrate the birthday of the Christ Child, and that the gifts customarily handed out symbolize the concepiton of Christ as God's greatest gift to the world.

This year it is particularly fitting that we remember and honor Him who has been called the Prince of Peace, and whose birth was hailed by the heavenly choir with the hymn, "Glory to God in the highest and on earth peace, good will toward men." The greatest Christmas gift that could come to this country and to the whole world would be that peace which He came to bring and which could be attained by the universal application of the Golden Rule.

WHY USE MORPHINE?

The time was when morphine was of necessity the drug of choice for relief of such severe pain as biliary or renal colic or the pain of a coronary occlusion. No other derivative of opium or any other analgesic which could be given by hypodermic injection could equal its power to relieve pain. It is no wonder that Sydenham said that without morphine few men would be callous enough to practice medicine, and that Osler called it "God's own medicine." The nausea that was apt to follow the administration of morphine was accepted as part of the price that had to be paid for the blessed relief it brought from pain; and this nausea, as well as its habit-forming property and the rapid tolerance acquired to it, was recognized as a calculated risk.

The custom of using morphine for any severe pain has become so firmly established that it may seem rank heresy to enter a protest against its use. In a treatise on cardiac therapy which is just off the press,[1] morphine is virtually the only drug recommended for the pain of myocardial infarction — although, as an apparent after-thought, the concession is made that "If patients cannot take morphine because of sensitivity, pantopon. . . or Demerol . . may be used as substitutes."[1]

Since morphine will nauseate at least one third—probably one half—of individuals not accustomed to its use, it seems pertinent to raise the question as to why it should be used at all, except for patients who are known to tolerate it well. Demerol or Dilaudid or methadone will relieve pain just as certainly and quickly as will morphine, and are far less apt to cause nausea than are morphine or Pantopon. In a patient with a myocardial infarction the violent exertion associated with vomiting may mean the difference between life and death.

Medical men should remember that there are substitutes for morphine which are just as effective and far safer. Why use morphine?

1. Stewart, H. J.: Cardiac Therapy, New York, Paul B. Hoeber, Inc., 1952, p. 305.

Clinicopathologic Conference

DUKE HOSPITAL

JAMES P. HENDRIX, M.D., *Editor*

Presentation of Case

DR. GRACE KERBY: This 34 year old housewife was apparently well until 50 days before her death when she developed cough, fever, generalized aching, and mild anorexia. She was admitted to her local hospital the next day with progressive abdominal distention, diffuse generalized abdominal pain and a temperature of 104 F. Terramycin therapy was instituted. Cough and fever disappeared, malaise and weakness persisted, abdominal distention increased, and nausea developed. After four days the liver was palpable and there was evidence of free fluid in the abdomen. The white blood cell count was 15,000; the icterus index was 12 units. Penicillin therapy was added. A normal bowel movement occurred on the ninth day. Abdominal distention persisted and increased. Blood nonprotein nitrogen was 70 mg. per 100 cc. Abdominal paracentesis on the tenth day yielded 200 cc. of clear amber fluid. The patient was transferred to Duke Hospital immediately after this procedure.

The past history is pertinent in that the patient had had abdominal distention and pain eight years previously, and an appendectomy was done. The liver was thought to be enlarged at that time. The patient manifested transient jaundice 10 to 14 days postoperatively and then ascites, necessitating repeated abdominal paracenteses. She appeared well after one to two months. There was no history of excessive alcohol intake, dietary inadequacy, toxic agents or of injections prior to the appendectomy. There was no history of jaundice in other members of the community at that time.

Physical examination on admission to Duke Hospital revealed a lethargic, somewhat dehydrated, acutely ill woman with no apparent icterus. Vital signs were normal except for slight tachycardia. There was a questionable spider angioma on the upper chest. Striking subcutaneous edema of the trunk was present below the level of the sixth to eighth thoracic vertebra. A few fine inspiratory rales were heard at both lung bases. The abdomen was tight, and there was evidence of free fluid. There was diffuse abdominal tenderness without definite rebound tenderness and without rigidity.

Laboratory findings: The hemoglobin was 12 Gm. and gradually fell during the next 40 days to 9.5 Gm. The white cell count varied between 13,000 and 41,000, with a polymorphonuclear leukocytosis. A few nucleated red blood cells were present peripherally. Urinalysis was normal. A stool examination revealed no occult blood. Blood nonprotein nitrogen was 42 mg. per 100 cc. The serum bilirubin never rose above 2.9 mg. per 100 cc. Serum amylase was 134 units. There was 38 per cent retention of bromsufalein dye at 45 minutes. Thymol turbidity was negative. Prothrombin was 23 per cent of normal. The serum proteins were 4.6 Gm. per 100 cc., with 2.4 Gm. of albumin and 1.2 Gm. of globulin. Blood cholesterol was 60 mg. per 100 cc. Serum electrolytes (milliequivalents per liter) were sodium 133, potassium 4, and chloride 92.

Course in the hospital: The patient remained essentially afebrile. Abdominal distention was persistent and unrelieved by usual measures. Peristaltic sounds were heard from time to time, and there were occasional spontaneous bowel movements. Abdominal pain was persistent and for the most part vague and diffuse, but worse after paracenteses. Ascites was marked and rapidly reaccumulative after paracenteses. The ascitic fluid was sterile, of low specific gravity, and no abnormal cells were seen on histologic examination of the sediment. After paracentesis 18 days before death, a tender, smooth liver edge was felt 3 fingerbreadths below the right costal margin, and a very tender 7 to 8 cm. epigastric mass, thought to be part of the liver, was palpated. After paracentesis on the day of death, the liver was no longer felt, and the left epigastric mass was thought to be definitely smaller than previously. The spleen was not felt at any time. An initial high protein, high calorie intake was subsequently changed to a low sodium diet, and mercurial diuretics were given. No diuresis was obtained, however. Thrombophlebitis developed in the right leg at the site of a venous cut-down, three days before death. The patient's course was gradually downhill, with a terminal semi-coma lasting several days. Two hours after the last paracentesis, the patient suffered a profound peripheral vascular collapse, from which she failed to recover, despite blood transfusions and other

supportive measures. She expired nine hours after the paracentesis.

Clinical Discussion

DR. KERBY: The most obvious finding in this case was the rapidly re-accumulating ascites in the absence of significant peripheral edema. The subcutaneous edema of the trunk probably resulted from subcutaneous leaking of ascitic fluid from the initial paracentesis wound. The character of the ascitic fluid and the lack of fever and of definite signs of peritoneal irritation make an inflammatory process unlikely. There was no evidence for metastatic tumor with peritoneal implants, although this cannot be ruled out because of failure to find tumor cells in the sedimented fluid. Portal hypertension seems the best probability, despite the lack of demonstrable splenomegaly. We know that the patient had hepatic decompensation eight years previously (jaundice and ascites). The history does not permit us to do more than speculate as to whether this episode represented an acute hepatitis or failure of a liver previously damaged by hepatitis or cirrhosis, and then further damaged by the stress of operation and anesthesia. We can assume, however, from the history, that our patient had previous liver damage as a background for the final illness. Our problem is whether the final illness was primary hepatic failure or whether a chronically damaged liver failed in the face of the superimposed burden of the acute febrile episode which ushered in the final illness. I favor the latter view because of the history of initial cough, fever and generalized aching, all of which responded promptly to antibiotic therapy; while the evidences of hepatic failure developed and progressed.

There are several features which raise the question of some additional factor in the final picture: (1) persistent, severe abdominal pain, worse after paracenteses; (2) persistent marked leukocytosis; and (3) the absence of jaundice as a conspicuous feature.

Neoplasm must be considered. There is no evidence for a primary metastatic tumor. Primary hepatoma is rare but occurs most commonly in cirrhosis, and may give a leukemoid blood picture and abdominal pain. The diffuse character of the pain and the decrease in size of the left epigastric mass are against this diagnosis.

Hepatic venous thrombosis may occur rarely with cirrhosis, producing abdominal pain, little jaundice, and rapidly recurring ascites. The absence of evident venous collateral circulation is against hepatic venous thrombosis in the present case.

Portal venous thrombosis is a less rare condition than hepatic venous thrombosis and may occur secondary to cirrhosis. Again, jaundice may be inconspicuous, ascites rapidly re-accumulates, and the white cells count may be high. Classically, the patient has abdominal pain, splenomegaly (but in many reported cases demonstrable only on pathologic examination) and hematemesis. There was no hematemesis, but massive bleeding of some other type may have precipitated the final profound peripheral vascular collapse.

In summary, it seems most likely that the patient had chronic liver disease, the etiology not determinable by history, and that there may be evidence of more recent damage as a result of the superimposed burden of the acute febrile illness. It is probable that an additional factor is present, and in order of probability I would list (1) portal venous thrombosis and (2), despite its rarity, primary hepatoma.

In order of probability, the cause of the terminal episode may have been (1) hemorrhage from (a) esophageal varices or (b) trauma related to the final paracentesis; or (2) vascular accident, either (a) pulmonary embolus (known thrombophlebitis of the leg) or (b) mesenteric venous thrombosis secondary to extension from portal venous thrombosis.

Clinical Diagnoses

1. Chronic liver disease with hepatic failure and possibly thrombosis of the portal vein.

2. Shock due to hemorrhage from esophageal varices or from trauma related to paracentesis.

Discussion of Pathologic Findings

DR. OSCAR DUQUE: The most significant findings were in the liver. The organ weighed 17,000 Gm., was irregularly nodular and scarred, with the right and left lobes approximately the same size. The mass felt on palpation during the physical examination was a nodular proliferation of hepatic tissue, 10 cm. in diameter, located to the left of the hilum. The surface of the liver presented numerous small and large yellowish nodules.

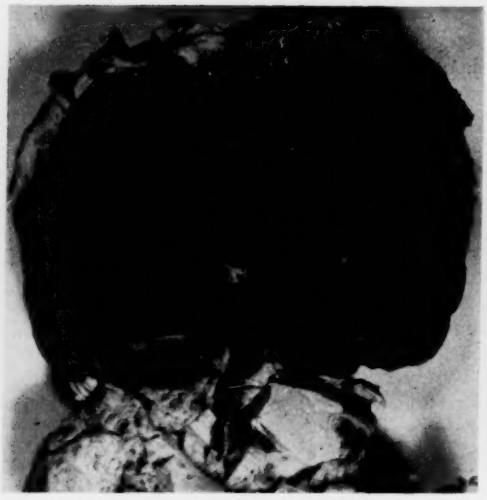

Fig. 1. Gross specimen. Horizontal section of the liver. Note the distorted shape of the organ and the bizarre appearance of the cut surface. Nodular areas of regeneration are prominent. Thrombosis of the portal vein may also be seen.

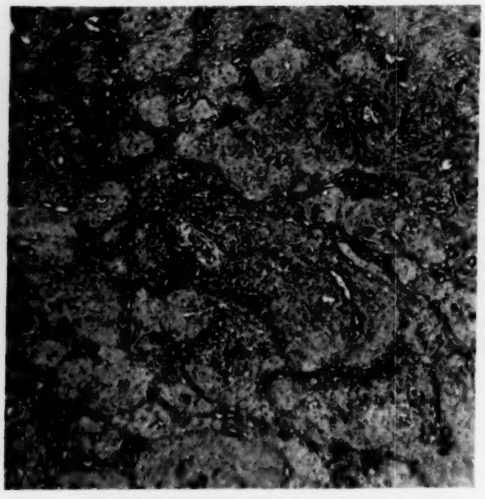

Fig. 2. Photomicrograph of an area of liver showing complete destruction of the parenchyma. The remaining lobular stroma appears demarcated by bile ducts and blood vessels.

The cut surface revealed a complete loss of the normal gross architecture (fig. 1). Bands of connective tissue divided the liver into irregular nodular lobules and areas of dark collapsed parenchyma. The appearance was that of the scarred liver of postnecrotic cirrhosis. There was thrombosis of the portal vein and its intrahepatic branches. In the trunk of the portal vein, the thrombosis was of long standing, since a new channel had formed. Thrombosis of the veins within the liver appeared to be quite recent.

Microscopically, the normal structure of the liver had been replaced by a combination of nodular hyperplasia, fibrosis, and hemorrhage. In large areas, the destruction of liver cells was complete, and the only remnants of hepatic structure appeared as collapsed lobular stroma demarcated by bile ducts and blood vessels (fig. 2). Nodules of regenerating liver cells were scattered throughout the liver. Discrete focal collections of inflammatory cells were also present.

The spleen was enlarged (700 Gm.), congested, and slightly fibrotic. There was a small area of splenic infarction. In the pancreas there were focal discrete areas of pancreatitis with fat necrosis.

To explain the disease process in this patient, one must consider first the nature of the hepatic lesion. The gross and microscopic appearance of the organ was that of the postnecrotic scarred liver that develops following destruction of the hepatic parenchyma by toxic or infectious agents. In the history of this patient there was an episode of jaundice, fever, and abdominal distention lasting from four to five weeks, eight years prior to her death. The anatomic findings in the liver suggest that this episode was an attack of the hepatocellular form of viral hepatitis. On the other hand, the symptoms of that episode may have been related to thrombosis of the portal vein, which in turn could be related to her appendectomy. However, portal thrombosis would have resulted in a different type of hepatic damage. Whatever the nature of the injury, the patient recovered, but her liver was left with a narrow margin of functional efficiency. The final acute liver failure was perhaps brought out by a new thrombosis of the portal system. The cause of this thrombosis is not known. One might attribute it to the slowing of the portal flow within the liver or perhaps to the mild pancreatitis or to both. Postnecrotic fibrosis of the liver is usually accompanied by splenomegaly, ascites, profound alteration of the protein metabolism with decrease in serum albumin, and late jaundice. All these changes were present in this case.

The immediate cause of death was mas-

sive intraperitoneal hemorrhage. During the last paracentesis the trocar had perforated a mass of large omental veins attached to the abdominal wall. Increased pressure in these veins and the bleeding tendency that occurs often in severe liver damage favored a profuse hemorrhage which might not have occurred in the absence of liver disease.

In summary, we have here the unusual case of a patient dying with a nodular postnecrotic fibrosis of the liver, eight years after an episode of jaundice. The degree of liver injury was such that this patient could never have recovered. The hemoperitoneum, following her last paracentesis, only hastened the final fatal outcome.

Anatomic Diagnosis

Severe post-necrotic fibrosis of liver (probably secondary to viral hepatitis). Old and recent thrombosis of portal vein. Chronic passive congestion, fibrosis and infarction of spleen. Adhesions between omentum and anterior abdominal wall. Traumatic rupture of omental veins by trocar (paracentesis). *Massive intraperitoneal hemorrhage.* Jaundice. Ascites.

New Desserts Remove Excess Salt

At the recent convention of the American Medical Association, visiting physicians made thousands of taste tests of cookies into which there had been baked a mixture of unflavored and finely ground sodium-removing resins. The doctors approved the medicated cookies for taste; many believed they compared favorably with grandma's best. Cookies are but one foodstuff into which the housewife may blend this new form of an already established agent which removes excess sodium from the bodies of patients who suffer from heart disease, cirrhosis of the liver, edema of pregnancy, or hypertension. Fruit juices, milk, fudge, cake, and flavored gelatin are some of the other nutriments which will hold and disguise the new medication. Now patients with excessive sodium retention can actually enjoy taking a medicine which allows other items in their diet to be seasoned more liberally and tastefully with once-forbidden salt. Eli Lilly and Company will supply physicians with books of various kitchen-tested recipes which may be given to patients.

Following its debut at the A.M.A. convention, 'Carbo - Resin' (Sodium Removing Resins, Lilly), Unflavored, has been placed on the prescription market.

Among the causes of hearing loss listed by the American Hearing Society are complications following measles, mumps, scarlet fever, chicken pox, whooping cough, meningitis; diseased tonsils; neglected, running ears; wax or foreign objects in ears; swimming in uninspected pools; blows on the ears; frequent colds.

BULLETIN BOARD

News Notes from the Duke University School of Medicine

Dr. Guy L. Odom, Duke University neurosurgeon, addressed the Montreal Neurosurgical Society in Montreal, Canada, November 12, on "Intracranial Bleeding of Non-Traumatic Origin." He also led a discussion on fractures of the spine, as well as participating in a neuropathology conference.

Dr. Odom served as visiting lecturer to students at McGill University Medical School, Montreal, November 10-15. With the medical students at McGill he discussed neurosurgical procedures for relief of pain.

Dr. Odom is past vice president of the Southern Neurosurgical Society.

* * *

Twenty Duke University doctors participated in the Southern Medical Association's annual meeting at Miami, Florida, November 10-13.

The Duke Medical School was represented in 11 of the 21 sections of the association, which embraces every phase of medical practice.

Duke physicians who delivered papers or led discussions were Dr. Charles W. Styron, Dr. R. W. Rundles, Dr. William N. Nicholson, Dr. W. B. Barton, Dr. E. Clinton Dexter, Jr., Dr. Julian M. Ruffin, Dr. John V. Kinross-Wright, Dr. Leslie B. Hohman, Dr. George A. Silver, Dr. George J. Race, Dr. J. Lamar Callaway, Dr. O. C. Hansen-Pruss, Dr. Susan C. Dees, Dr. J. Leonard Goldner, Dr. John Glasson, Dr. Walter L. Thomas, Dr. Robert N. Creadick, Dr. Robert L. Alter, Dr. Ralph A. Arnold, and Dr. George J. Baylin.

Among the scientific exhibits was one by Dr. J. E. Markee, chairman of Duke's Anatomy Department, on "The Autonomic Nervous System."

* * *

Dr. Edwin P. Alyea, Duke University urologist, was recently elected president of the North Carolina Urological Association for 1952-53.

Dr. Alyea, in charge of the Duke Urologic Division, succeeds Dr. William Lott of Asheville.

Other new officers are Dr. John Rhodes of Raleigh, vice president; and Dr. Louis C. Roberts of Durham, secretary-treasurer. Dr. Roberts is also a member of the Duke medical staff.

The 50-member Association holds annual scientific and business sessions. The next regular fall meeting will be held at Southern Pines in 1953.

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Dr. Arthur F. Abt, director of the Radioisotope Unit of the new Veterans' Administration Hospital in Durham, has been appointed a professor of pediatrics at Duke University Medical School, Dean W. C. Davison announced recently.

Dr. Abt is one of seven chiefs of services who will conduct the medical program of the new 500-bed hospital which is expected to open in Durham about April 1.

Dr. Abt has been associated with the Radioisotope Unit at Hines, Illinois, since last March and acting chief, Research Division, Research and Education Service, Department of Medicine and Surgery of the VA, Washington, D. C., since July.

A diplomate of the American Board of Pediatrics, he has been a practicing pediatrician since 1926.

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A $2,000 Easter Seal grant will aid in the expansion of Duke University's speech service to North Carolinians, it was announced recently.

The North Carolina State Society for Crippled

Children and Adults has made the grant to Dr. Leslie B. Hohman, director of the Duke Child Guidance Clinic, for treatment of speech disorders by Dr. Murray M. Halfond, medical speech pathologist at Duke Hospital and at the North Carolina Cerebral Palsy Hospital. The grant will make possible the treatment of speech handicapped children and adults who otherwise could not afford to pay, Dr. Hohman said.

Application for diagnosis of children's speech difficulties will be accepted at the Duke Child Guidance Center. Adults should apply directly to Dr. Halfond, director of Medical Speech Pathology, Duke Hospital.

The new service is part of an expanding program in cooperation with state agencies to reduce the number of North Carolina's speech problems and to develop better treatment methods, Dr. Halfond said.

* * *

Duke University Medical School has just acquired a picture collection of famous physicians and medical scenes for the new Duke Medical Research Building, Dean W. C. Davison announced recently. Purchase of the prized collection has been made available through a grant from the Doris Duke Foundation. The 250 framed steel engravings have been collected over a long period by Dr. J. Calvin Weaver of Atlanta, Georgia, an authority on Georgia medical history.

Duke Medical School Library houses the library of the Georgia Medical Society founded in 1804 at Savannah.

News Notes from the University of North Carolina School of Medicine

Dr. James C. Andrews, professor and chairman of the Department of Biological Chemistry and Nutrition, has received a grant of nearly $6,000 from the U. S. Public Health Service to support a program of research on the production of calcium oxalate stones and other stones in kidney and urinary bladder. The frequent occurrence of such stones, often requiring surgical removal, makes this study one of especial interest. This area of the state has often been described as a "stone area" because of the frequency with which such stones occur. The present work, for which the U. S. Public Health Service has offered support, was initiated some years ago at the suggestion of Dr. George L. Carrington of the Alamance General Hospital, Burlington. Dr. Carrington very generously furnished financial support for several years' research in this department. At that time the work was largely concerned with the oxalate content of common foods of this region. This oxalate, from food sources, can contribute materially to the formation of oxalate stones in some individuals. The new grant will enable a continued program of research to be carried on in cooperation with the Memorial Hospital and the School of Dentistry. To the dentist, the formation of "pulp stones" is a matter of considerable interest. Members of the dental faculty are cooperating with Dr. Andrews and his co-workers in a study of possible conditions which might be a cause of both pulp stones and renal stones.

* * *

Mr. Milton Huppert, of the Department of Bacteriology, has received a grant from the Pharma-Craft Corporation of New York City in the amount of $1,822 for continuation of studies on a project aimed at developing increased resistance against superficial fungus infection by immunization techniques.

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Dr. Edward C. Curnen, professor of pediatrics, met with the Central Ohio Pediatric Society in Columbus in December and discussed "Major Viruses and Minor Illnesses."

* * *

Dr. Ernest Craige. assistant professor of medicine, addressed the New England Heart Association on "The Relation of the Electrocardiogram to Red Blood Cell Chemical Changes in Diabetic Acidosis" at its annual meeting in Boston. Dr. Louis G. Welt, also of the Department of Medicine, presented papers on "Fluid and Electrolyte Metabolism" and "The Pathogenesis of Dehydration" before the Veterans Administration Teaching Hospital staff in Memphis the latter part of November.

* * *

Dr. K. M. Brinkhous, professor of pathology, attended the second Cold Injury Conference of the Josiah Macy, Jr. Foundation in New York on November 20 and 21. Dr. C. Bruce Taylor, of the Department of Pathology, attended the annual meeting of the American Society for the Study of Arteriosclerosis in Chicago; he read a paper entitled "Factors Affecting the Distribution of Vascular Disease in Hypercholesterolemic Rabbits." Dr. George D. Penick attended the meeting of the Southern Surgical Association and Southeastern Section of the American College of Pathologists in Miami in November.

* * *

Members of the Department of Surgery participated in several meetings and conferences during November: Dr. Nathan A. Womack attended meetings of the Council of the American Surgical Association in Cleveland, the Surgical Committee of the National Research Council in Washington, and the Seaboard Medical Association; at the latter meeting he spoke on "The Relation of Benign Lesions of the Breast to Cancer." Dr. Paul Bunce was also a guest speaker at the Seaboard Medical Association meeting; his topic was "Cancer of the Prostate." Dr. Colin G. Thomas spoke to the U. S. Naval Hospital staff at Camp Lejeune on "Injuries of the Hand."

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Dr. John T. Sessions, of the Department of Medicine, met with the Rocky Mount Academy of Medicine on November 20; he discussed "The Clinical Significance of Some Benign Gastrointestinal Lesions."

Public Relations Conference

The sixth annual Public Relations Conference of the State Medical Society was held Wednesday, December 17, at the Sir Walter Hotel in Raleigh.

Speakers for this year's conference were Philip G. Rozelle, Southeastern Director of Public Relations for General Motors Corporation, Atlantic; Robert J. Putnam, Industrial and Community Relations Department, Champion Paper and Fiber Company, Canton; and W. F. DeLoache, Southeastern regional manager, Public Relations Department, E. I. Du Pont de Nemours and Company, Charlotte. Also invited was Thurman Sensing, executive vice president of the Southern States Industrial Council, Nashville.

Attending the conference were state, district, and county society officers, public relations committee members, councilors, and Auxiliary officials.

North Carolina Mental Hygiene Society

"Our Mentally Handicapped Children" was the theme of the Mental Hygiene Institute held in Raleigh on November 14, under the sponsorship of the North Carolina Mental Hygiene Society and the North Carolina Mental Health Council. Dr. David A. Young, superintendent, Hospitals Board of Control, gave a statement of the present situation, and Dr. Roger Howell, professor of mental health of the University of North Carolina School of Public Health, led a discussion on "What Can We Do About It?"

Dr. W. Carson Ryan of the Department of Education, University of North Carolina, was moderator for a panel of speakers composed of psychologists, educators, public welfare workers, psychiatrists and psychiatric case workers, law enforcement officers, and a juvenile court judge.

North Carolina Alcoholic Rehabilitation Program

One of the greatest morale builders offered to the patients at the North Carolina Butner Alcoholic Rehabilitation Center has been the bi-weekly visits of a representative from the North Carolina Employment Service in Durham, who interviews the men with an eye to their proper employment when they leave Butner. It has long been recognized that the sense of inadequacy and anxiety peculiar to the man who cannot find work in keeping with his abilities, or who cannot properly support his family, is often a major contributing factor in the illness of alcoholism. A man working hard, in a congenial atmosphere, at a job he enjoys, with an adequate salary and an appreciative employer, is seldom tempted to drown his worries in drink.

In March of 1951, Mr. S. K. Proctor, executive director of the North Carolina Alcoholic Rehabilitation Program, secured the services of the Durham Employment Office in interviewing the patients, registering them, and forwarding their cards to the areas of their choice. The men also receive information about available jobs, either in their home towns or in other localities, if they desire a change, about unemployment compensation, on-the-job training, and guidance and counsel relative to vocational choice and work attitudes. All employment offices in the state are cooperating in the preparation of the patient to assume financial responsibility upon his discharge from Butner.

According to Director Proctor, during the last two months there has been a change in the Durham Employment Office's services to Butner patients. Because of the demand for such service and the success of the program, Mr. C. B. Sessoms, trained counselor and interviewer, visits the Center every Friday afternoon, thereby getting the opportunity to talk to each patient two or three times during the patient's 28-day stay at Butner. Sessoms' aid was made possible through the efforts of Mrs. Ethel Carr Lipscomb, manager of the Durham Employment Office.

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Horace M. Champion of Raleigh has joined the staff of the North Carolina Alcoholic Rehabilitation Program as public information officer, executive director S. K. Proctor announced recently. Champion, who succeeds Santford Martin, Jr., in this capacity, will edit the ARP publications, including **Inventory**, North Carolina's bi-monthly journal on alcohol and problems of alcoholism.

North Carolina Heart Association

A community service program, sponsored by the Cabarrus County Heart Association, has been established in Cabarrus County. The program functions through a monthly clinic called the Cardiac Rehabilitation Clinic. Patients are seen only by referral from their physicians. The clinic offers services which are ordinarily beyond the scope of the physician-patient relationship, such as: vocational rehabilitation through contact with the State Vocational Rehabilitation office; methods of conserving energy in housework ("Heart of the Home"); dietetic advice by dietitians; qualified recreational outlets through local community recreation commission facilities; and home nursing supervision. In addition, the clinic will consider any other problem for which the physician may wish to send his patient. Not all patients can be benefited, of course, but it is felt that an indefinite number of services can be offered, are offered only if the need exists, and are offered with maximum efficiency of time and money.

* * *

Scientific meetings on cardiovascular subjects, sponsored by the Durham-Orange County Heart Association, are again being held monthly. The November meeting was held on November 11, in the auditorium of North Carolina Memorial Hospital; Dr. Lewis Welt spoke on "Electrolyte Abnormalities in Congestive Heart Failure." The next meeting was on December 9, at 8:00 p. m., at Duke Hospital, when Dr. J. C. Meyers discussed "Shock." Co-chairmen of the Scientific Meetings Committee are Dr. Bernard Holland of Durham and Dr. Carl Gottschalk of Chapel Hill.

* * *

The North Carolina Heart Association's new Committee on Rheumatic Fever held its first meeting on November 23, in Greensboro, to discuss short and long-range programs for exploring and combating the rheumatic fever problem in the state. Dr. E. P. Benbow of Greensboro, chairman of the committee, presided. Twenty-one members of the committee, representing various agencies and groups concerned with the problem, attended the session.

Halifax County Medical Society

The Halifax County Medical Society held its regular monthly dinner and meeting at the Roanoke Rapids Hospital on November 14. Dr. James F. Donnelly of the Bowman Gray Medical School faculty, Winston-Salem, spoke on "Maternal Mortality."

Edgecombe-Nash Medical Society

Dr. R. A. Ross of the University of North Carolina School of Medicine was speaker at the monthly meeting of the Edgecombe-Nash Medical Society on November 12.

Carteret County Medical Society

The Carteret County Medical Society held its monthly meeting November 10, at the Morehead City Hospital. This was a dinner meeting, the hospital acting as host.

The scientific program consisted of a paper by Dr. William H. Bell, radiologist, for the Morehead City Hospital. Dr. Bell presented a number of interesting x-ray plates showing aneurysm of the aorta, achondroplasia, a benign bone cyst of the humerus, and neurogenic bladder with bladder stones, following traumatic injury to the lumbar spine.

Dr. N. Thomas Ennett, chairman of the Public Relations Committee of the society, discussed the seventh annual National Essay Contest for high school students sponsored by the Association of

American Physicians and Surgeons. At a previous meeting the local society decided to sponsor this contest for the Carteret County high schools.

The subject of this contest is "Why the Private Practice of Medicine Furnishes this Country with the Finest Medical Care." The first prize on the national level is $1,000.00, and the first prize on the local level is $25.00. The contest begins January 1, and closes March 1.

Dr. M. B. Morey, president, presided.

N. THOS. ENNETT, M.D.
Corresponding Secretary

MEDICAL DEPARTMENT, ECUSTA PAPER CORPORATION

On December 2 the Medical Department of the Ecusta Paper Corporation, Pisgah Forest, North Carolina, will conduct a meeting for the Regional Chapter of the Society for the Advancement of Management. The theme of the meeting will be "The Value of Medical Maintenance for Industrial Workers."

Dr. Mac Roy Gasque, medical director of Ecusta Paper Corporation, Pisgah Forest, North Carolina, will be the general chairman of the medical part of the program. Others appearing on the program are: William H. Seymour, vice president, Liberty Mutual Insurance Company, Boston, Massachusetts; Dr. Manson Meads, Department of Preventive Medicine, Bowman Gray School of Medicine, Winston-Salem; Dr. H. Charles Ballou, the Greenbrier Clinic, White Sulphur Springs, West Virginia; Dr. Allan J. Fleming, assistant medical director, E. I. du Pont de Nemours & Company, Wilmington, Delaware; Dr. Edward C. Holmblad, managing director, Industrial Medical Association, Chicago, Illinois.

NEWS NOTES

Dr. Julius A. Howell has announced the opening of offices in Winston-Salem for the practice of otorhynolaryngology and maxillofacial surgery.

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Dr. E. W. Larkin, Jr., was certified by the American Board of Ophthalmology following the examination in October.

EMORY UNIVERSITY SCHOOL OF MEDICINE

The Woodruff Memorial building, $2,000,000 center for medical research in the Southeast, was opened recently at Emory University. The seven-story structure is connected by closed walkways with Emory University hospital, and was made possible by grants from private donors and from the National Cancer Institute.

Emory medical school research in such fields as biochemistry, pathology, anatomy, neurosurgery, thoracic surgery, and use of radioisotopes, will be extended, and scientists from several areas will work together on related projects, according to Dr. R. Hugh Wood, dean of the medical school.

The building was begun in 1950, and has been named for the late Ernest Woodruff, pioneer Southern industrialist, who established the Woodruff Foundation. Funds from this Foundation were contributed to the new research facilities at Emory.

"The new building will serve the Southeast and the entire nation, and will add impetus to the development of Atlanta as a great medical center," said Dean Wood.

NEWS NOTES FROM THE AMERICAN MEDICAL ASSOCIATION

Improving health services of our nation's working force will be the over-all theme of the Annual Congress on Industrial Health, sponsored by the A.M.A.'s Council on Industrial Health. Workers, industrial leaders and medical men will assemble for this thirteenth annual conference January 21-22 at the Drake Hotel, Chicago.

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Today's Health magazine is taking its place with regular textbooks on the college campus. More than 1,300 freshman physical education students at the University of Illinois at Champaign-Urbana are reading the magazine as part of their regular classroom assignment. A special supplementary study sheet called "Classroom Discussion Topics" is being supplied free of charge by the A.M.A.'s Bureau of Health Education as an aid to classroom discussion.

* * *

American Medical Education Foundation state chairmen will kick off the 1953 fund-raising drive with a meeting Sunday, January 25, at the Sheraton Hotel in Chicago. This second annual meeting will launch officially the medical profession's concerted effort during the coming year to raise voluntary funds to assist the country's medical schools. Keynote speaker will be L. D. McGuire. M.D., A.M.E.F. chairman for Nebraska.

* * *

A new pamphlet—"The AMAzing Story"—prepared by the American Medical Association, gives the general public a brief summary of the ways in which the A.M.A. serves an average American family. This booklet pictorializes the many ways the Association serves Mr. and Mrs. Joe Typical and family.

* * *

Medical societies which are anticipating displaying health exhibits at local fairs and meetings during the coming year, will be interested in the attractive new exhibit catalogue prepared by the A.M.A.'s Bureau of Exhibits. This booklet carries pictures of 25 different health exhibits, with brief descriptions and such installation data as the minimum space and electrical requirements and shipping weights. All of these exhibits are available on a loan basis from the Bureau. The booklets are being mailed to secretaries of state medical associations for distribution to county societies.

* * *

The 1952 edition of "Apparatus Accepted," a publication of the A.M.A.'s Council on Physical Medicine and Rehabilitation, is now available. The 60-page booklet includes a complete listing of diagnostic and therapeutic devices which have been accepted by the Council. Among other things, the booklet lists such items as audiometers and hearing aids, electrocardiographs, oxygen therapy apparatus, incubators, inhalers and resuscitators, and metabolism testing equipment. Copies may be secured on request through the Council.

* * *

The story of how local medical societies bring medical care and medical services to more and more American people is the subject of a new exhibit which is nearing completion by the A.M.A.'s Bureau of Exhibits. "You and Your Medical Care" exhibit features emergency call services, voluntary health insurance, early detection and prevention of such diseases as cancer and TB, community health councils, grievance committees, and sources of health education information.

A separate unit has been designed for each subject, making the exhibit suitable for showings in every community . . . any portion of the exhibit

may be omitted if a state or county society has not yet developed a program on a certain subject. The exhibit will be available shortly after the first of the year.

* * *

More and more Americans will have an opportunity to see the RKO-Pathe film, "Your Doctor," during 1953. An estimated 12 million persons viewed the 15-minute movie-short in more than 2,000 commercial theaters across the nation in 1952.

After January 1, 16 mm. prints of the film will be available for showings to schools, service clubs, civic groups, industrial plants and special professional meetings. Produced by Louis de Rochemont in cooperation with the A.M.A., this film portrays the work of 35 year old Dr. George Bond in his valley clinic in North Carolina.

The American Medical Association also has made arrangements to obtain prints for state and county society film libraries. Societies interested in purchasing a print at cost may write to the A.M.A. Public Relations Department. Details on distribution methods will be made available later.

* * *

"Health for Today," an authoritative health column by Dr. W. W. Bauer, director of the A.M.A.'s Bureau of Health Education, will be syndicated six days a week from coast-to-coast through King Features Syndicate. The first article will appear January 5, 1953. In his column, Dr. Bauer will discuss mainly health subjects rather than disease. He has received authorization from the Board of Trustees.

American College of Surgeons

An impressive program of symposiums, panel discussions, clinical conferences, and medical motion pictures on practical surgical problems will open the 1953 season of Sectional Meetings of the American College of Surgeons at The Netherlands Plaza Hotel in Cincinnati, Ohio, January 19-21. This meeting is the first of eight scheduled for various parts of North and South America during the coming year.

Other sectional meetings will be held this year in Atlanta, Georgia, February 23-24; Boston, Massachusetts, March 2-5; Salt Lake City, Utah, March 20-21; Oklahoma City, Oklahoma, March 24-25; Los Angeles, California, March 30-31; Calgary, Alberta, April 23-24. The first Inter-American Session will be held in Sao Paulo, Brazil, February 9-12.

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Ceremonies marking the end of a 35-year period in which the American College of Surgeons held sole responsibility for setting standards for the nation's hospitals will be held Saturday, December 6, when the plan will be transferred to the new Joint Commission on Accreditation of Hospitals in a program at the John B. Murphy Auditorium, Chicago, at 4 p.m.

Principal speaker will be Senator Lister B. Hill of Alabama, co-sponsor of the Hill-Burton Act, instrumental in constructing over 1,000 hospitals, especially in rural communities. Dr. Evarts A. Graham, chairman of the Board of Regents of the College, will turn the new program over to Dr. Gunnar Gundersen, first chairman of the Commission.

From now on the American College of Physicians, American College of Surgeons, American Hospital Association, American Medical Association, and Canadian Medical Association will share responsibilities of the program. Proposal for the formation of the commission was initiated by the College in the recognition that not only surgeons but other members of organizations concerned with medical care in hospitals should participate in the establishment and enforcement of hospital standards. This combination of organizations constitutes an important milestone in the development of health services for the American people.

American Academy of Dermatology and Syphilology

The eleventh annual meeting of the American Academy of Dermatology and Syphilology was held December 6-11 in the Palmer House, Chicago.

In addition to the many papers presented, there were several symposiums and panel discussions. Officers of the Academy this year are: Dr. C. Guy Lane, Boston, president; Dr. Michael H. Ebert, Chicago, vice president; Dr. John E. Rauschkolb, Cleveland, secretary-treasurer; and Dr. James R. Webster, Chicago, assistant secretary-treasurer.

National Society for Crippled Children and Adults

Appointment of three new members to the professional staff of the National Society for Crippled Children and Adults includes Fletcher C. Kettle as director of organization and administration, Donald C. Morris as managing editor of publications, and Mrs. Lydia Newton as program consultant.

Kettle, who will supervise the business management of the nationwide Easter Seal Society, formerly served for six years as president of Larry Burns Motors, Inc., Chicago. Prior to this he was deputy director of the Division of Displaced Persons of the United Nations Relief and Rehabilitation in London, England, and was chief liaison officer with General Eisenhower's headquarters.

Morris will serve as managing editor of the **Crippled Child Magazine** and **Bulletin**, official publications of the Easter Seal Society. He comes to the National Society from the University of Chicago, where he was editor of the University of Chicago Magazine, which, under his editorship last summer, received the national Magazine-of-the-Year award of the American Alumni Council.

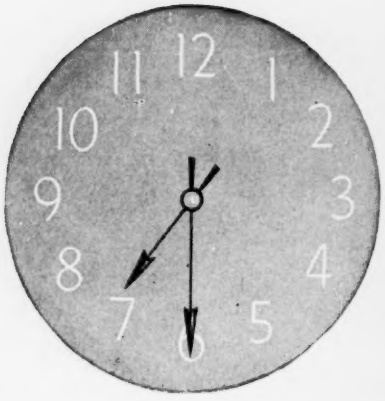
Mrs. Newton's duties will involve working with the more than 2,000 state and local Easter Seal societies throughout the nation in extending treatment and care for crippled children. A graduate of Northwestern University, she was formerly director of the speech and hearing program for the Arizona Society for Crippled Children and Adults in Phoenix.

American Diabetes Association

The first postgraduate course in diabetes and basic metabolic problems to be conducted by the American Diabetes Association will be offered under the direction of Dr. Charles H. Best, director of the Banting and Best Department of Medical Research of the University of Toronto, on January 19, 20, 21, 1953, at the University of Toronto, Canada.

Developed by the Association's Committee on Postgraduate Education, under the chairmanship of Dr. Edward L. Bortz, the course will have as its clinical director, Ray F. Farquharson, M.B., professor of medicine of the University of Toronto, and Andrew L. Chute, M.D., professor of pediatrics of the University of Toronto, will act as associate clinical director.

Over thirty lectures and round-table discussions have been planned. The course is open to non-member physicians as well as members of the American Diabetes Association, but the number of registrants will be limited to 100. Fees are $20 to members, $40 to non-members. Details of the three-day program and registration and hotel information may be obtained from J. Richard Connelly, Executive Director, American Diabetes Association, 11 West 42nd Street, New York 36, N. Y.



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BOOK REVIEWS

Cardiac Therapy. By Harold J. Stewart, M.D., Associate Professor of Medicine, Cornell University Medical College; Attending Physician, New York Hospital; Head of Division of Cardiology, Department of Medicine, New York Hospital-Cornell Medical Center. 622 pages. Price, $10.00. New York: Paul B. Hoeber, Inc., 1952.

In this book Dr. Stewart gives his readers the benefit of a professional lifetime of experience in treating diseases of the heart and in teaching successive generations of students. It is one of the most comprehensive books on cardiac therapy yet published, and is one of the best. It is well written, practical, and gives in detail the author's methods of treating all the known ills the heart is heir to. It can be heartily recommended to internists and general practitioners who want to know the last word in cardiac therapy.

One of the best, if not the best, chapters in the book is chapter 32—"What to Tell Patients About Their Heart Disease."

About the only criticism this reviewer would offer is that Dr. Stewart follows the time-honored custom of prescribing morphine for the relief of pain in myocardial infarction. The impression is left that this is by all odds the drug of choice. Since morphine nauseates most people and since the nausea and attendant vomiting may prove dangerous or even fatal, there seems little excuse nowadays for not substituting Demerol, Dilaudid, or methadone, which are effective pain-relievers and much more easily tolerated. This minor criticism, however, is not intended to detract from the value of the book as a whole.

Cardiography in General Practice. Electrocardiography, Vectorcardiography and Ballistocardiography. By Abraham I. Schaffer, M.D., Assistant Visiting Physician, Metropolitan City Hospital, New York. 13 pages. Price, $3.00. Baltimore, Maryland: The Williams and Wilkins Company, 1952.

According to its title, this text is proposed for the use of the general practitioner of medicine.

The section covering electrocardiography might be of some value to one with some training in this field. However, the portions of the work dealing with vectorcardiography and ballistocardiography are far beyond the reach of all general practitioners and the majority of those who are already trained in the ordinary use of the electrocardiograph.

Furthermore, the uses of these methods have been presented as the "final court of appeal" in many cases of cardiovascular disease. Any internist recognizes the value of the electrocardiograph but puts greater faith in a well taken history and careful physical examination.

The statement is made that leads V-2 and V-5, along with the conventional limb leads, are "sufficient for screening purposes." However, it is well known that the American Heart Association's standard screening chest position is CF-4.

For the foregoing reasons, this text is far from ideal for the general practitioner.

Correlative Neuroanatomy and Functional Neurology. By Joseph J. McDonald, M.D., and Joseph G. Chusid, M.D. Ed. 6. 261 pages. Price, $4.00. Los Altos, California: Lange Medical Publications, 1952.

This concise volume, in outline form, presents in a simple manner the essential features of the anatomy and physiology of the nervous system and relates these aspects of the subject to the field of clinical neurology.

Although this book is designed for the beginner in neurology, it should be of value to medical students, postgraduate students and physicians, either in general practice or in various specialties, who desire a review of the subject of neurology and all its branches. Although it does not replace the standard textbooks of neuroanatomy, neurophysiology, neuropathology, or neurology, it may serve as a handy reference to the busy student or physician.

The first two sections of the book are devoted to the central and peripheral nervous system respectively. The third section is devoted to the principles of neurodiagnosis. This section includes a useful guide to neurologic examinations, including a precise outline of neurologic signs and symptoms.

Brain Surgeon. The Autobiography of Dr. William Sharpe. 271 pages. Price, $3.75. New York: The Viking Press, 1952.

This is a rather dramatic attempt on the part of the author to portray his life to the public and to put forth certain theories the author has regarding the prevention and treatment of cerebral palsy and the treatment of hydrocephalus. His theories are so many years behind the newer advances in neurophysiology and neuropathology that this book will scarcely raise any comment from those interested in the field of neurology in this regard. The author grew up in a period when neurosurgeons were few and far between, and could have achieved great fame in the field of neurosurgery alone. He is, however, hardly known by the younger generation of neurosurgeons.

There are quite a few interesting anecdotes written along the line of consultation room stories giving the author's point of view toward many of the great men of his earlier years. This book will make an interesting evening of reading for those who desire to learn more about the author but will not leave the lasting impression of Dr. John Fulton's biography of Harvey Cushing.

Nutrition and Diet in Health and Disease. By James S. McLester, M.D., Professor of Medicine Emeritus, University of Alabama; and William J. Darby, M.D., Ph.D., Professor of Biochemistry and Director of the Division of Nutrition, Vanderbilt University. Ed. 6. 710 pages. Price, $10.00. Philadelphia and London: W. B. Saunders Company, 1952.

The sixth edition of this outstanding book represents an extension, revision, and amplification of the previous volume. Biochemical diagnostic measures to re-evaluate requirements have been utilized to bring up to date the data on food composition.

Approximately one third of the book concerns itself with diet and food factors, and an analysis and physiologic evaluation of major food products, vitamins, and minerals. Special consideration is given to infant-feeding and diet during pregnancy and lactation.

The remaining two thirds of the book discuss the application of diet, not only to the production of disease, but in the management of dietary, metabolic, infectious, and degenerative disorders. Topics of current interest include diets for hypertension, renal disease, and functional and organic digestive disturbances, as well as problems presented by patients pre- and post-operatively.

The comprehensive nature of the material offered in this single volume, in consideration of the tremendous expansion of the subject since the fifth edition, would not have been possible without the critical selectivity evident in the authors' work.

This book remains one of the most valuable contributions in a field which touches every aspect of medicine. It is highly recommended to all students and practitioners of medicine.

Standard Values in Blood: Being the first fascicle of a Handbook of Biological Data. Prepared under the direction of the Committee on the Handbook of Biological Data, American Institute of Biological Sciences, The National Research Council. Edited by Errett C. Albritton, M.D., Fry Professor of Physiology, George Washington University. Ed. 6. 199 pages. Price, $4.50. Philadelphia and London: W. B. Saunders Company, 1952.

This is an enormously detailed compilation of data on blood, the work of over 600 people, and is the first fascicle of a handbook of biological data. It is almost Teutonic in scope and conception. The material is discussed under the following headings: physical properties, coagulation phenomena, blood groups, erythrocytes and hemoglobin, leukocytes, blood and bone marrow cells, water, carbohydrate, lipid, protein, amino acids, non-protein nitrogen, phosphorus, sulfur, vitamins, hormones, enzymes, electrolytes, minerals, gases, acid-base, effects of readiation, storage, therapeutic agents. There is a bibliography and index. The format is photo-offset, and the cover is composition.

To review this work adequately would require the efforts of a panel such as the one which produced it. Really, one can only announce it. In scanning the sections which deal with the reviewer's field of interest, he finds surprising omissions in such a welter of data. In the section on the bone marrow there is no information on the degree of cellularity of the marrow at different ages, as seen in sections. This information has been available for 20 years, and appears in standard reference works such as Wintrobe's text and Custer's Atlas. The latter work, although in extensive use in this country, does not anpear in the bibliography. If this is any indication of the state of affairs elsewhere, it may be that even this great compilation is incomplete. Nonetheless, the information assembled here is of great value, and will save many people hours of paging through various publications.

Culdoscopy: A New Technic in Gynecologic and Obstetric Diagnosis. By Albert Decker, M.D., F.A.C.S., Clinical Professor of Gynecology and Obstetrics, New York Polyclinic Medical School and Hospital. 148 pages, with 50 figures. Price, $3.50. Philadelphia and London: W. B. Saunders Company, 1952.

For the occasional gynecologic or obstetric problem when direct visualization of the pelvic viscera may be required for accurate diagnosis, the relatively simple approach through the cul-de-sac employing a suitable telescopic instrument may replace a more hazardous laparotomy.

The author introduced celioscopy by the vaginal route (culdoscopy) and improved the technique by placing the unanesthetized subject in the knee-chest position and developed a suitable instrument (the Decker culdoscope) for this approach.

In this monograph the author describes his technique and reviews his experience over the past seven years. Culdoscopy aids primarily in the diagnosis of ectopic pregnancy, endometriosis, ill-defined pelvic masses, and infertility. The complications of culdoscopy are few; contraindications are infrequent. This complete guide will be welcomed by interested gynecologists and surgeons.

For Boys Only. By Frank Howard Richardson, M.D. 91 pages. Price, $2.75. Atlanta, Georgia: Tupper and Love, Inc., 1952.

This reviewer has read many books discussing the question of sex and advising the older generation how to tell the facts of life to the younger generation. The best one that he has yet seen for adolescent boys and their parents is the one by Frank Howard Richardson. Dr. Richardson is a pediatrician who has learned a good deal about children the hard way—by dealing with four of his own, three sons and one daughter.

The book discusses in simple, everyday terms and in conversational style virtually all of the so-called "facts of life" that teen-age boys need to know. While written, as the name implies, for boys only, it could be read with profit by the parents of adolescent boys. It is a book which can safely be recommended for placing in the hands of any intelligent teen-age boy.

In Memoriam

JAMES GILLIAM JOHNSTON, M.D.
(1872—1952)

With the passing of Dr. James G. Johnston to richer fields of service and to a greater reward, this Society of his fellow members is deeply conscious of the loss of a worthy colleague, and a true and amiable friend.

He was possessed of a fine mind which gave him the capacity to think clearly and to arrive at logical conclusions. Because of his modesty he never put these faculties on display except to his most intimate confreres. He had a quiet, keen sense of humor, and a demeanor which never seemed to be disturbed even under the most trying circumstances. He was a man and a physician of the highest integrity and ethical principles, and a Christian gentleman. No one could ever say of him that he ever made a derogatory statement about any man. His willing and untiring efforts to help his fellow man, his faith and belief, and love of human kind, and the Christian spirit through which he conducted every phase of his daily life will serve as a living monument to him which all of us should be mindful of and should strive to emulate.

"For when The One Great Scorer
comes to write your name;
He writes—Not that you won or lost,
but how you played the game."

James Gilliam Johnston's creed of living was conducted according to those concepts. His attainments and accomplishments as a physician, a civic leader, and a churchman need not be recorded here. They are indelibly written in the record of his life, and will always remain fresh in the memories of his colleagues, his patients and his friends.

We, his brother physicians and all others who knew him, will miss his wise counsel, and gentle guiding hand.

Be It Resolved: That The Mecklenburg County Medical Society record its feelings of high tribute to him as a physician, confrere, and friend, and our deepest sympathy to his bereaved family and,

Be It Further Resolved: That a copy of these resolutions be included in the permanent records of this Society, and a copy be sent to his family and to the **North Carolina Medical Journal.**

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*Hamilton, H. E., and Zavala, D. C.: Amebiasis in Iowa: Diagnosis and Treatment, J. Iowa M. Soc. 42:1 (Jan.) 1952.

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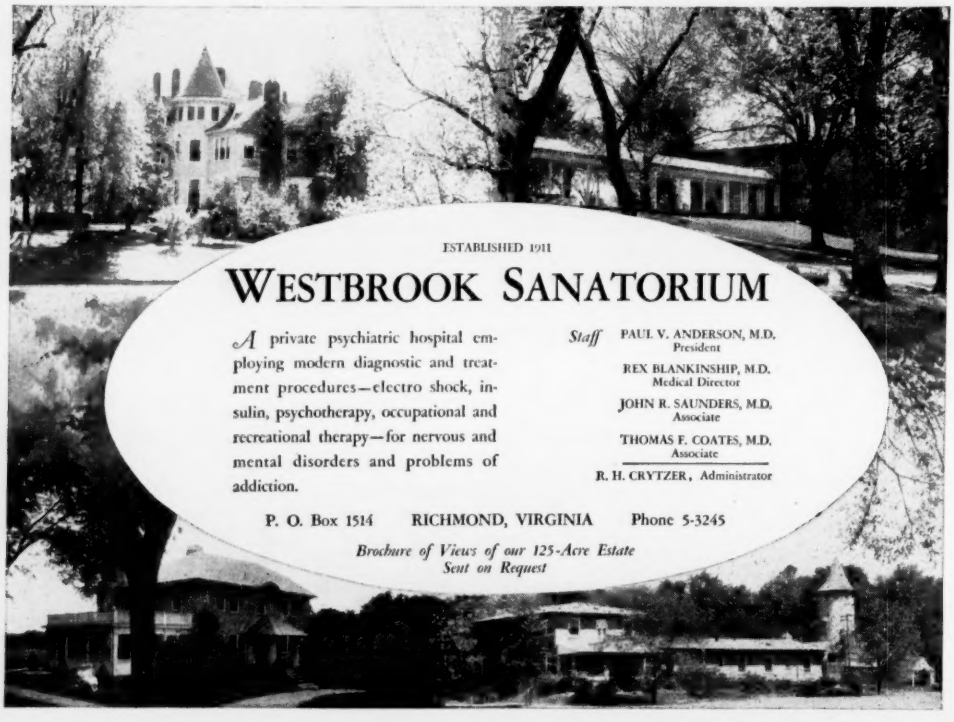

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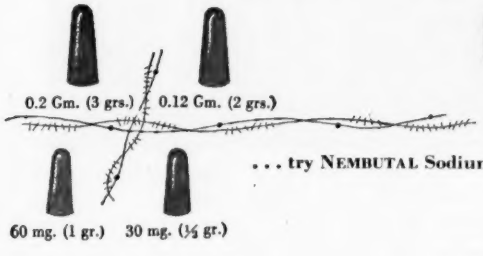

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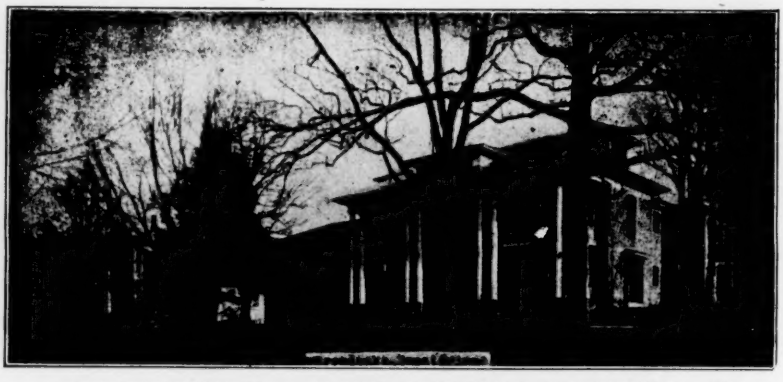

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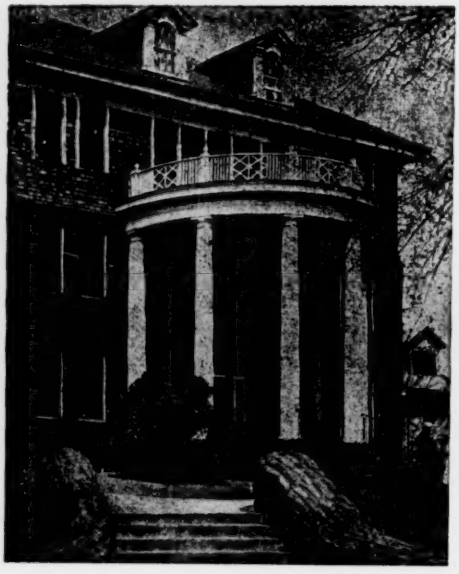

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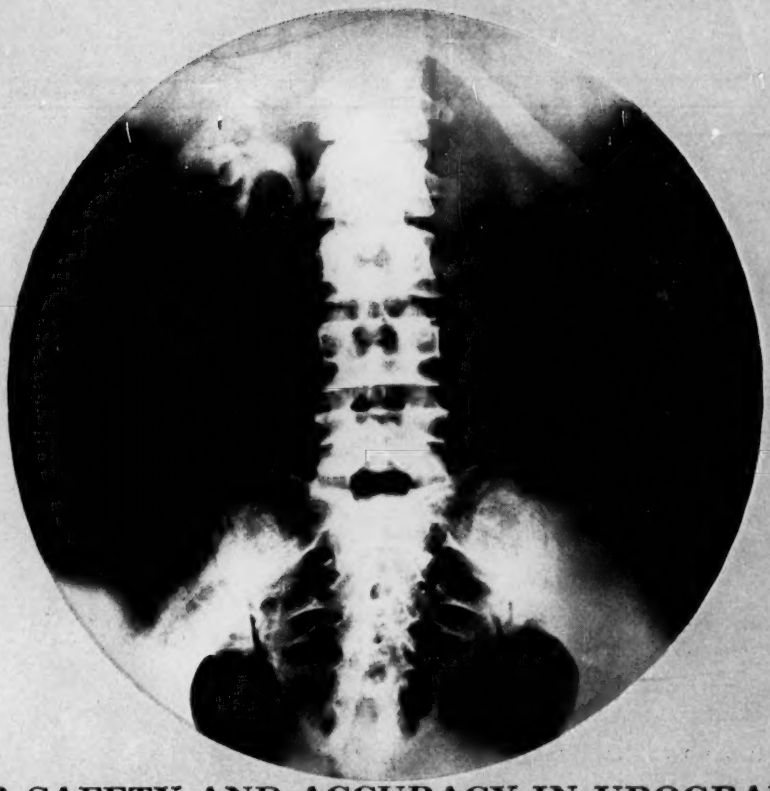

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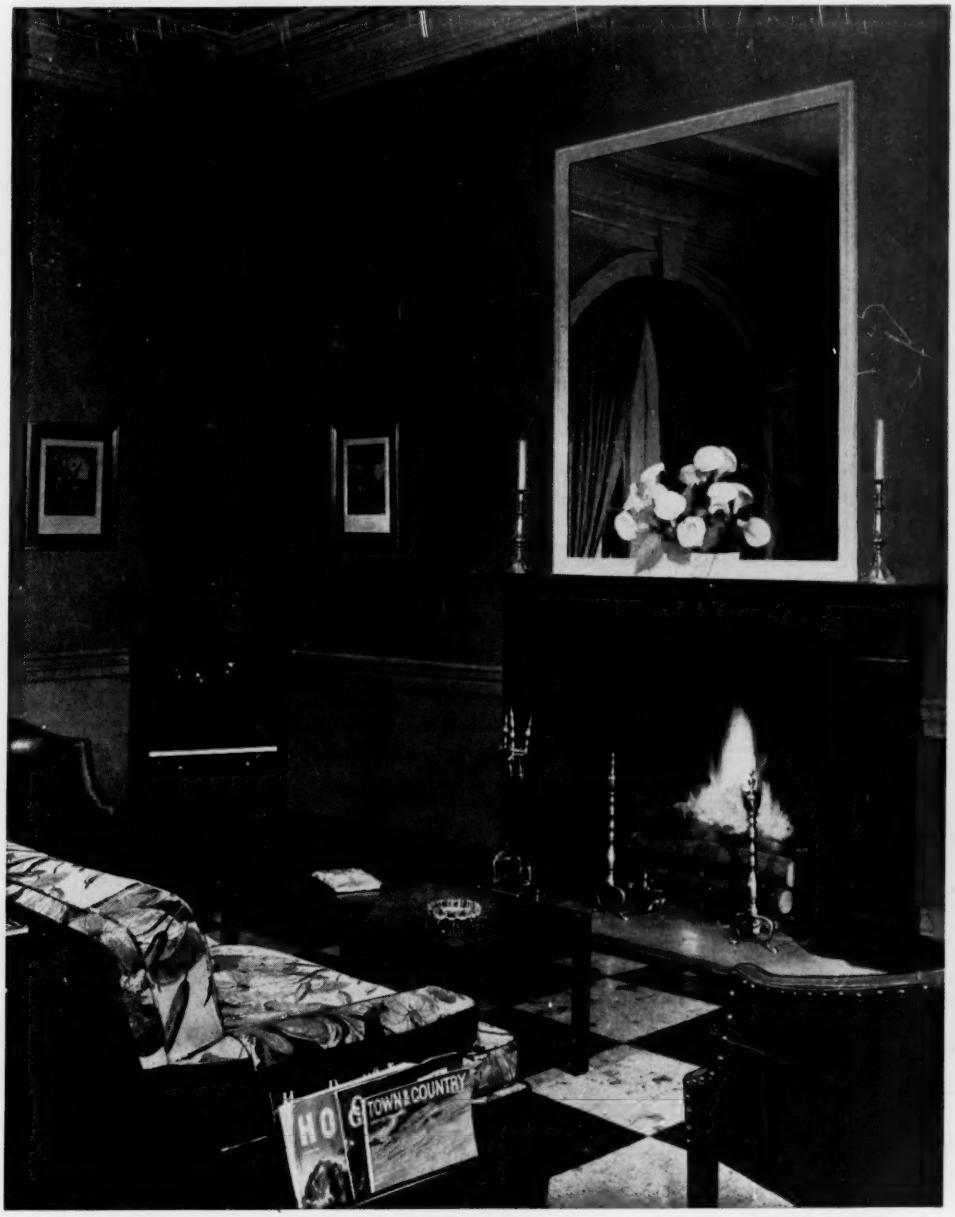

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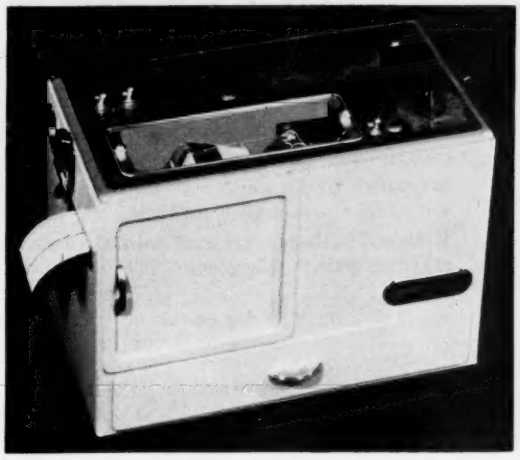

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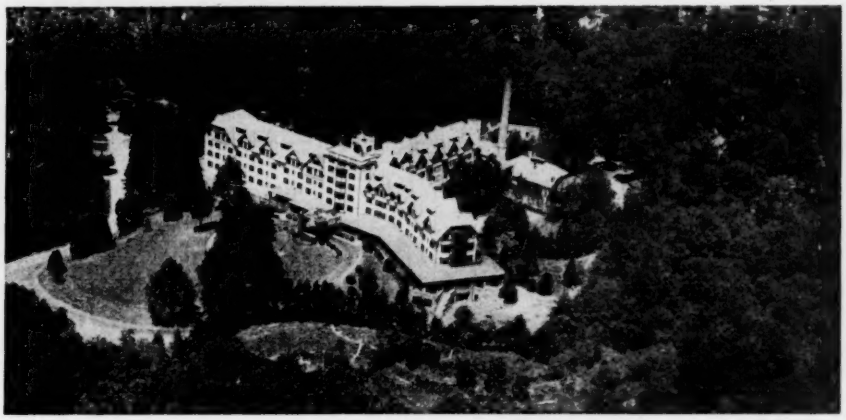

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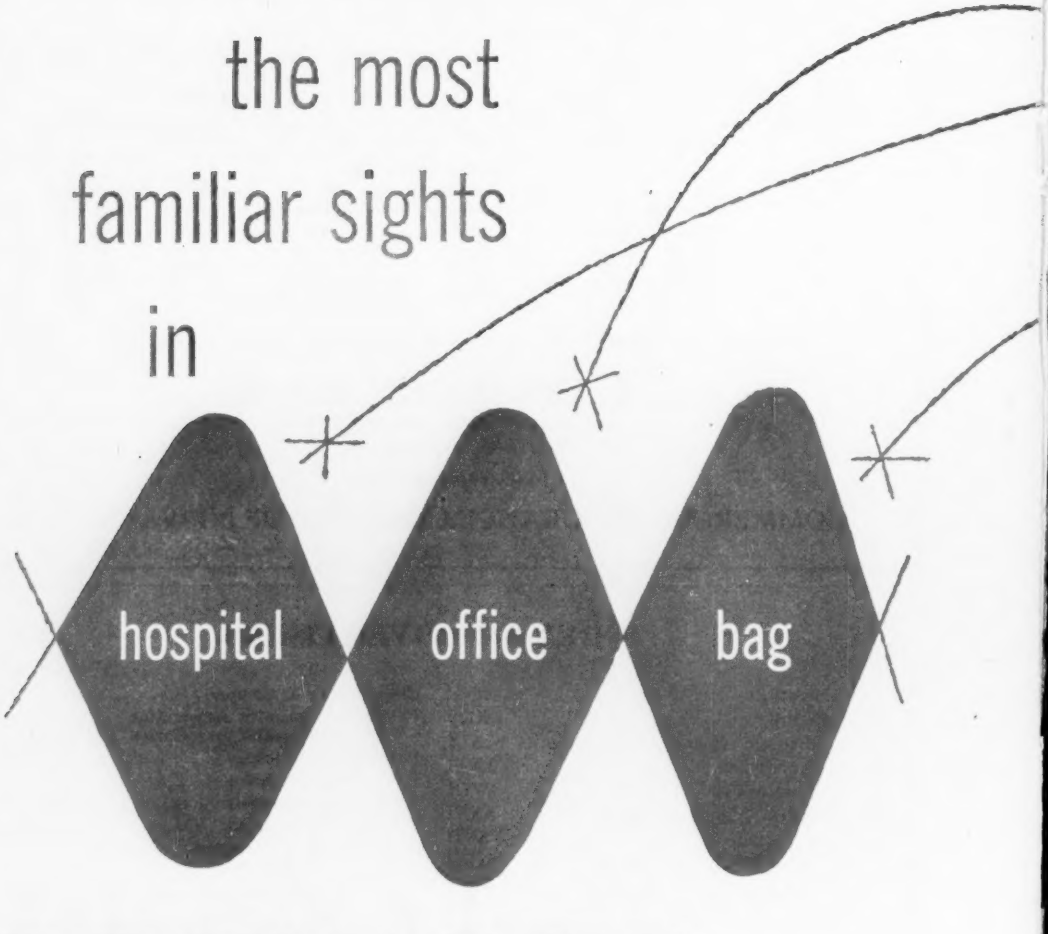

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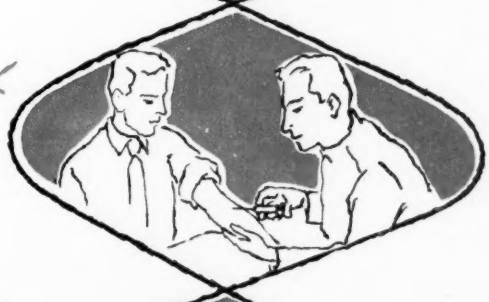

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